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Issue Date: 07 June 2006

In the Matter of

NORMA J. PARSONS, Widow of, and on behalf of,
BILLY J. PARSONS
Claimant

v.

WESTMORELAND COAL COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party in Interest

Case No.: 2004 BLA 14
2004 BLA 15

Appearances: Mr. Ron Carson, Lay Representative
For the Claimant

Ms. Mary Rich Maloy, Attorney
Mr. Christopher H. Hunter, Attorney (on brief)
For the Employer

Before: Richard T. Stansell-Gamm
Administrative Law Judge

DECISION AND ORDER –
DENIAL OF BLACK LUNG DISABILITY BENEFITS CLAIM, and
DENIAL OF MODIFICATION REQUEST IN SURVIVOR CLAIM

This matter involves a claim filed by Mr. Billy J. Parsons for disability benefits and a claim by Mrs. Norma J. Parsons for survivor benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 ("the Act"), as implemented by 20 C.F.R. Parts 718 and 725. Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as "black lung" disease.

Procedural Background and Discussion

Mr. Parsons' Black Lung Disability Claim

First Claim
(DX 47)[1]

On October 28, 1971, Mr. Parsons applied for federal black lung disability benefits with the United States Department of Health, Education and Welfare. In September 1973, the Social Security Administration denied the claim. However, following the 1977 amendments to the Act, the United States Department of Labor ("DOL") reviewed the claim under the new entitlement standards. Eventually, the Deputy Commissioner issued a Decision and Order, dated December 7, 1982, awarding black lung disability benefits contingent upon Mr. Parsons' retirement from coal mine employment within one year. Mr. Parsons never responded to a letter from DOL concerning future employment plans. The official record indicates that Mr. Parsons did not retire for Westmoreland Coal Company until March 1985.

Second Claim

On February 24, 1986, Mr. Parsons completed a second application for federal black lung disability benefits. On May 22, 1987, a DOL claims examiner denied benefits because Mr. Parsons failed to establish pneumoconiosis arising out of coal mine employment or total respiratory disability. On June 1, 1987, Mr. Parsons appealed, requesting a formal hearing before the Office of Administrative Law Judges ("OALJ"). After conducting a formal hearing in Abingdon, Virginia on May 22, 1990, Administrative Law Judge Charles Campbell credited Mr. Parsons with twenty-seven years of coal mine employment and identified Westmoreland Coal Company as the proper responsible operator. On the merits, Judge Campbell concluded that Mr. Parsons established pneumoconiosis through x-ray evidence pursuant to 20 C.F.R. § 718.202(a)(1), but failed to establish total respiratory disability pursuant to 20 C.F.R. § 718.204(b). Accordingly, Judge Campbell denied benefits.

On January 2, 1991, Mr. Parsons appealed to the Benefits Review Board ("BRB" and "Board"). However, on April 28, 1992, with the appeal still pending, Mr. Parsons proffered additional medical evidence with a request for modification. Accordingly, the BRB remanded his claim to the District Director for adjudication of the modification. On April 16, 1993, DOL denied the modification request and Mr. Parsons appealed, requesting another formal hearing. In response, the District Director in June 1993 conducted an informal conference in Norton, Virginia to clarify issues before forwarding the claim to OALJ. However, when benefits were still denied, Mr. Parsons in August 1993 again appealed the adverse decision. After conducting a hearing in July 1994, Administrative Law Judge Reno Bonfanti, by Decision and Order dated September 30, 1994, denied Mr. Parsons' modification request, concluding Mr. Parsons failed to establish a mistake of fact or a material change in conditions pursuant to 20 C.F.R. § 725.309. Specifically, Judge Bonfanti did not disturb Judge Campbell's finding that Mr. Parsons had pneumoconiosis which arose out of coal mine employment. At the same time, Judge Bonfanti

[1]The following notations appear in this decision to identify exhibits: DX – Director exhibit; CX – Claimant exhibit; EX – Employer exhibit; ALJ – Administrative Law Judge exhibit; and TR – Transcript.

found insufficient evidence to establish total disability. On October 17, 1994, Mr. Parsons appealed the denial of his claim to the BRB. On April 24, 1995, the BRB dismissed Mr. Parsons' appeal for failure to file a Petition for Review and an accompanying legal brief.

Third, and Present, Claim

On July 24, 1996, Mr. Parsons filed his third application for benefits under the Act. DOL adjudicated the application as a duplicate claim under 20 C.F.R. § 725.309 (DX 1). On April 14, 1997, the District Director denied Mr. Parsons' duplicate claim for failure to establish pneumoconiosis arising out of coal mine employment, total respiratory disability or a material change in conditions (DX 30). On April 21, 1997, Mr. Parsons requested a reconsideration and additional time to procure chest x-ray interpretations (DX 33). In May 1997, the District Director once again denied Mr. Parsons' application for federal black lung disability benefits for failure to establish pneumoconiosis arising out of coal mine employment, total respiratory disability or a material change in conditions pursuant to 20 C.F.R. § 725.309 (DX 36). On May 23, 1997, Mr. Parsons appealed, requesting a formal hearing. DOL referred the case to OALJ in August 1997. On October 30, 1998, I issued a Decision and Order, awarding benefits to Mr. Parsons. At that time, I determined Mr. Parsons had established that he suffered a totally disabling pulmonary impairment and correspondingly a material change in conditions. Upon consideration of the entire record and applying collateral estoppel, I determined that Mr. Parsons was totally disabled due to coal workers' pneumoconiosis (DX 64). On November 30, 1998, the Employer appealed the award of benefits (DX 65).

On November 30, 1999, the BRB affirmed in part and vacated in part my determinations and remanded the case to me for further adjudication (DX 74). In its remand order, the BRB affirmed the following determinations: a) Westmoreland Coal Company is the responsible operator; b) Mr. Parsons was not able to establish total disability under 20 C.F.R. §§ 718.204 (c) (1) – (3) and 718.304; c) Mr. Parsons established a totally disabling respiratory impairment through the preponderance of medical opinion under 20 C.F.R. § 718.204 (c) 4); and Mr. Parsons established a material change in conditions under 20 C.F.R. § 725.309(d). However, the BRB rejected my application of *res judicata* and collateral estoppel to establish the presence of pneumoconiosis and pneumoconiosis arising out of coal mine employment under 20 C.F.R. §§ 718.202 (a) (1) and 718.203 (b). The Board also vacated my finding that the opinions of physicians who failed to diagnose pneumoconiosis should be discredited under 20 C.F.R. § 718.204 (b).

On May 21, 2001, upon discovery that Mr. Parsons had passed away and autopsy evidence of pneumoconiosis existed, I remanded his claim to the District Director for further consideration (DX 78). On June 17, 2003, the District Director denied Mr. Parsons' claim for benefits because he had failed to prove that he was totally disabled due to coal workers' pneumoconiosis (DX 82). Mrs. Parsons, on behalf of her deceased husband, appealed and requested a hearing with OALJ (DX. 85). The District Director returned the case to OALJ on October 8, 2003 (DX 186). After several continuances, and pursuant to a Notice of Hearing, dated November 24, 2004, (ALJ I), I conducted a hearing on March 17, 2005 in Abingdon, Virginia. Mrs. Parsons, Mr. Carson, and Ms. Maloy were present at the hearing. My decision in

the case is based on the hearing testimony and the following documents admitted into evidence: DX 1 to DX 187, CX 1, and EX 1 to EX 6.

Discussion

Close review of the BRB's November 1999 order is necessary to establish the present procedural stance of Mr. Parsons' third claim. Specifically, the Board remanded the case to me to re-adjudicate three issues: the presence of pneumoconiosis, pneumoconiosis arising out of coal mine employment, and total disability due to pneumoconiosis. However, significantly, the BRB also affirmed my determination that Mr. Parsons has established a material change in conditions by demonstrating that he had become totally disabled due to respiratory problems since the denial of his most recent, second claim in April 24, 1995. Based on that affirmation, under the provision of 20 C.F.R. § 725.309 (d) (pre-2001),[2] denial of his third claim is no longer warranted and I must consider the entire record to determine whether he was totally disabled due to coal workers' pneumoconiosis.[3]

Mrs. Parsons' Survivor Claim

On May 3, 1999, Mrs. Parsons filed a claim for survivor benefits under the Act (DX 91). The District Director denied the claim on February 3, 2000 (DX 129). Following Mrs. Parsons' March 10, 2000 appeal, the case was forwarded to OALJ on May 26, 2000 (DX 131 and DX 144). On October 18, 2000, Administrative Law Judge Mollie Neal conducted a hearing (DX 155). On September 26, 2001, Judge Neal denied Mrs. Parsons' survivor claim because the more probative medical opinion failed to establish that Mr. Parsons' death was due to coal workers' pneumoconiosis (DX 164). Mrs. Parsons appealed on October 8, 2001 (DX 165). On June 28, 2002, the BRB affirmed Judge Neal's denial of benefits (DX 171). On June 17, 2003, Mrs. Parsons requested modification of the denial decision and submitted an additional medical opinion (DX 172). The District Director denied her modification request on August 11, 2003; Mrs. Parsons appealed on August 22, 2003; and, the case was forwarded to OALJ on October 8, 2003 (DX 176, DX 179, and DX 183). Again, on March 17, 2005, I conducted a hearing with the parties present. Likewise, my decision on Mrs. Parsons' modification request is based on the hearing testimony and the following documents admitted into evidence: DX 1 to DX 187, CX 1, and EX 1 to EX 6.

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[2]In January 2001, a new set of DOL regulations concerning the adjudication of black lung claims became effective. Most of the new regulations in 20 C.F.R. Part 718 and some portions of Part 725 are applicable to Mr. and Mrs. Parsons pending claims. However, the old version of 20 C.F.R. § 725.309, which I have designated with the suffix "(pre-2001)," controls the procedural status of Mr. Parsons' present claim. *See* 20 C.F.R. § 725.2 (c).

[3]In his closing brief, Employer's counsel raises several concerns with my 1998 determination concerning respective probative weight of the medical opinions. In particular, he believes subsequent case law: a) diminishes the probative weight that may be placed on the opinions of treating physicians; and, b) enhances the probative weight of opinions by board certified physicians. While I will address those concerns during my determination of the entire record, I believe the 1999 BRB affirmation of my finding of a material change in conditions has become the law of the case, which in turn warrants consideration of the entire record on entitlement to benefits rather than reconsideration of the material change in conditions issue.

ISSUES

Mr. Parsons' Black Lung Disability Claim

Whether Mr. Parsons is entitled to black lung disability benefits under the Act.

Mrs. Parsons' Survivor Claim

1. Whether in filing a modification request on May 13, 2003, Mrs. Parsons has demonstrated a mistake in determination of fact occurred in Director's denial of his most recent prior claim on May 14, 2002.

2. If Mrs. Parsons establishes a mistake in determination of fact, whether coal workers' pneumoconiosis caused or contributed to the death of her husband, Mr. Billy Parsons.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Preliminary Findings

Born on July 14, 1935, Mr. Parsons married Mrs. Norma J. Parsons on September 24, 1956. Mr. Parsons passed away on June 2, 1998. In June 1954, Mr. Parsons began coal mine employment for Mason Coal Company. After working with eight small coal companies as a battery motor operator, shuttle car operator and hand loader, Mr. Parsons found employment with Westmoreland Coal Company on February 2, 1968 as an insider laborer. In seventeen years as an employee of Westmoreland Coal Company at Big Stone Gap, Virginia, Mr. Parsons labored as a continuous mining machine operator and a shuttle car operator (DX 47, DX 91, DX 96, DX 97, and TR, pages 14-19).[4]

Stipulations of Fact

At the March 17, 2005 hearing, the parties stipulated that: a) Mr. Parsons had at least 18 years of coal mine employment; b) Westmoreland Coal Company is the responsible operator; c) Mrs. Norma J. Parsons is an eligible dependent for the purposes of augmentation of disability benefits that may be payable under the Act; d) Mrs. Norma J. Parsons is an eligible survivor under the Act; and, e) Mr. Billy J. Parsons had coal workers' pneumoconiosis. (TR, pages 8, 9, 19 and 20).

Mr. Parsons' Black Lung Disability Claim

To establish entitlement to black lung disability benefits under Act, a claimant must prove by a preponderance of the evidence four elements of entitlement. First, the miner must

[4]The location where the claimant last engaged in coal mine employment determines which federal Court of Appeals has appellate jurisdiction. *Shupe v. Director, OWCP,* 12 B.L.R. 1-200, 1-202 (1989)(en banc). In this case, the US Court of Appeals for the Fourth Circuit has jurisdiction.

establish the presence of pneumoconiosis.[5] Second, if a determination has been made that a miner has pneumoconiosis, it must be determined whether the miner's pneumoconiosis arose, at least in part, out of coal mine employment.[6] Third, the miner has to demonstrate he is totally disabled.[7] And fourth, the miner must prove the total disability is due to pneumoconiosis.[8]

Pneumoconiosis

"Pneumoconiosis" is defined as a chronic dust disease arising out of coal mine employment.[9] The regulatory definitions include both clinical or medical, pneumoconiosis, defined as diseases recognized by the medical community as pneumoconiosis, and legal pneumoconiosis, defined as "any chronic lung disease arising out of coal mine employment."[10] The regulation further indicates that a lung disease arising out of coal mine employment includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."[11] As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

As noted the above, the Employer stipulated that Mr. Billy J. Parsons had coal workers' pneumoconiosis. Based on that stipulation and noting that the autopsy/pathology evidence in this case establishes the presence of pneumoconiosis under 20 C.F.R. § 718.202 (a) (2), I find Mr. Parsons had pneumoconiosis.

Pneumoconiosis Arising Out of Coal Mine Employment

Once a Claimant has proven the existence of pneumoconiosis, 20 C.F.R. § 718.203 (a) requires that he also establish that his pneumoconiosis arose at least in part from his coal mine employment. According to 20 C.F.R. § 718.203 (b), if the Claimant was employed in coal mining for ten or more years, a rebuttable presumption that the pneumoconiosis is due to coal mine employment exists.

Again, at the March 2005 hearing, the parties stipulated that Mr. Parsons had coal workers' pneumoconiosis and worked at least eighteen years as a coal miner. Based on their stipulations, pathological evidence of coal workers' pneumoconiosis, while considering the causation presumption under 20 C.F.R. § 718.203 (b) and in the absence of sufficient contrary evidence, I find Mr. Parsons' pneumoconiosis arose out of his coal mine employment.

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[5]20 C.F.R. § 718.202.

[6]20 C.F.R. § 718.203 (a).

[7]20 C.F.R. § 718.204 (b).

[8]20 C.F.R. § 718.204 (a).

[9]20 C.F.R. § 718.201 (a).

[10]20 C.F.R. §§ 718.201 (a)(1) and (2).

[11] 20 C.F.R. § 718 (b).

Total Disability

To receive black lung disability benefits under the Act, a claimant must have a total disability due to a respiratory impairment or pulmonary disease. If a coal miner suffers from complicated pneumoconiosis, there is an irrebuttable presumption of total disability. 20 C.F.R. §§ 718.204 (b) and 718.304. If that presumption does not apply, then according to the provisions of 20 C.F.R. §§718.204 (b) (1) and (2), in the absence of contrary evidence, total disability in a miner's claim may be established by four methods: (i) pulmonary function tests; (ii) arterial blood-gas tests; (iii) a showing of cor pulmonale with right-sided, congestive heart failure; or (iv) a reasoned medical opinion demonstrating a coal miner, due to his pulmonary condition, is unable to return to his usual coal mine employment or engage in similar employment in the immediate area requiring similar skills.

While evaluating evidence regarding total disability, an administrative law judge must be cognizant of the fact that the total disability must be respiratory or pulmonary in nature. In *Beatty v. Danri Corporation. & Triangle Enterprises and Director., OWCP*, 49 F.3d 993 (3d Cir. 1995), the court held that in order to establish total disability due to pneumoconiosis, a miner must first prove that he suffers from a respiratory impairment that is totally disabling separate and apart from other non-respiratory conditions.

The record does not contain sufficient evidence to establish that Mr. Parsons suffered cor pulmonale with right-sided congestive heart failure. As a result, total respiratory or pulmonary disability must be established through the presence of complicated pneumoconiosis, pulmonary function tests, arterial blood gas studies, or medical opinion.

Complicated Pneumoconiosis

The regulation, in part, at 20 C.F.R. § 718.304, provides that if a claimant is able to establish the presence of complicated pneumoconiosis, then an irrebuttable presumption of total disability and death due to pneumoconiosis is established. In the Black Lung Benefits Act, 30 U.S.C. 921 (c) (3) (A) and (C), as implemented by 20 C.F.R. § 718.304 (a), Congress determined that if a miner suffered from a chronic dust disease of the lung which "when diagnosed by chest X-ray, yields one or more large opacities (greater than one centimeter in diameter) and would be classified in category A, B, or C," there shall be an irrebuttable presumption that his death was due to pneumoconiosis.[12] This type of large opacity is called "complicated pneumoconiosis." The statute and regulation, 30 U.S.C. 921 (c) (3) (B) and (C) and 20 C.F.R. §§ 718.304 (b) and (c), also permit complicated pneumoconiosis to be established by either the presence of massive fibrosis in biopsy and autopsy evidence or other means which would be expected to produce equivalent results in chest x-rays or biopsy/autopsy evidence.

[12]On the standard ILO chest x-ray classification worksheet, Form CM 933, large opacities are characterized by three sizes, identified by letters. Category A indicates the presence of a large opacity having a diameter greater than 10 mm (one centimeter) but not more than 50 mm; or several large opacities, each greater than 10 mm but the diameter of the aggregate does not exceed 50 mm. Category B means an opacity, or opacities "larger or more numerous than Category A" whose combined area does not exceed the equivalent of the right upper zone of the lung. Category C represents one or more large opacities whose combined area exceeds the equivalent of the right upper zone.

According to the U.S. Court of Appeals for the Fourth Circuit in *Eastern Associated Coal Corp. v. Director, OWCP [Scarbro]*, 220 F.3d 250 (4th Cir. 2000), the existence of complicated pneumoconiosis is established by "congressionally defined criteria." As a result, the statute's definition of complicated pneumoconiosis as radiographic evidence of one or more large opacities categorized as size A, B, or C, 30 U.S.C. 921 (c) (3) (A), represents the most objective measure of the condition. This sets the benchmark by which other methods for proving complicated pneumoconiosis are measured, as described in 30 U.S.C. 921 (c) (3) (B) and (C). *Id*. at 256. In other words, whether a massive lesion or other diagnostic results represent complicated pneumoconiosis under 30 U.S.C. 921 (c) (3) (B) and (C) requires an equivalency evaluation with the x-ray criteria set forth in 30 U.S.C. 921 (c) (3) (A).[13] Additionally, the court emphasized that the legal definition of complicated pneumoconiosis as established by Congress controls over the medical community's definition of the disease. *Id*. at 257. Finally, the court indicated that although all relevant and conflicting medical evidence must be considered and evaluated:

> if the x-ray evidence vividly displays opacities exceeding one centimeter, its probative force is not reduced because the evidence under some other prong is inconclusive or less vivid. Instead, the x-ray evidence can lose force only if other evidence affirmatively shows that the opacities are not there or are not what they seem to be, perhaps because of an intervening pathology, some technical problem with equipment, or incompetence. *Id*.

Referencing a 1993 case from the U.S. Court of Appeals for the Fourth Circuit, *Lester v. Director, OWCP,* 993 F.2d 1143, 1145-46 (4th Cir. 1993) the Benefit Review Board recently emphasized in *Mullins v. Plowboy Coal Company,* No. 04-0716 BLA, Unpublished, July 8, 2005, that an administrative law judge "must weigh together all of the evidence relevant to the presence or absence of pneumoconiosis." That mandate is consistent with other case law indicating that all evidence relevant to whether the miner has pneumoconiosis must be weighed. *Gray v. SLC Coal Co*., 176 F.3d 382 (6th Cir. 1999), *Melnick v. Consolidation Coal Co.,* 16 B.L.R. 1-31 (1991); *Maypray v. Island Creek Coal Co.*, 7 B.L.R. 1-683 (1985).

In other words, even if the presence of large opacities has been established through one of the three methods set out in § 718.304, all other medical evidence must be considered and evaluated to determine whether the large opacities actually exist and represent complicated pneumoconiosis. For example, the Benefits Review Board affirmed a finding of complicated pneumoconiosis under 20 C.F.R. §718.304 when the administrative law judge considered chest x-rays in conjunction with CT-scan findings to determine there was sufficient evidence to find complicated pneumoconiosis. *Keene v. G&A Coal Co.*, BRB No. 96-1689 BLA (Sept. 27, 1996). And, in another case, despite radiographic evidence of large opacities, the U.S. Court of Appeals for the Sixth Circuit upheld a determination that complicated pneumoconiosis did not exist based on probative autopsy evidence indicating the lesions were not complicated pneumoconiosis. *Gray*, 176 F.3d at 388.

With these principles in mind, I turn to two specific chest x-rays in the extensive radiographic record which is summarized later in this decision. In the September 23, 1996 chest

[13]See also 20 C.F.R. §§ 718.304 (b) and (c).

x-ray, Dr. Wheeler, a dual qualified radiologist, identified a pulmonary mass between 1.5 cm to 2 cm mass (DX 31). Although Dr. Wheeler opined the mass was consistent with healed tuberculosis, it nevertheless might qualify as a Category A radiographic opacity which may represent the presence of complicated pneumoconiosis. However, upon consideration of other evidence in the record, I find Dr. Wheeler's radiographic finding does not establish the presence of complicated pneumoconiosis. Two subsequent CT scans, which identified a pulmonary mass did not lead to a diagnosis of complicated pneumoconiosis. More significantly, as discussed later, based on the autopsy of Mr. Parsons' lungs and microscopic examination of the lung tissue, none of pathologists or other physicians who reviewed the autopsy/biopsy results believed the pulmonary mass represented complicated pneumoconiosis. Instead, Dr. Naeye observed one conglomeration of silicotic nodules. Dr. Kahn reported one "confluence" of such nodules. Dr. Caffrey observed confluent nodules. While their observations may explain Dr. Wheeler's radiographic findings, Dr. Naeye, Dr. Kahn, and Dr. Caffrey did not diagnose the collection of silicotic nodules as complicated pneumoconiosis. Dr. Naeye also specifically explained why the conglomeration was not complicated pneumoconiosis.

In the chest x-ray, dated February 19, 1997, Dr. Mathur, a dual qualified radiologist, observed size A large opacity (DX 43 and DX 46), which raises the possibility of complicated pneumoconiosis. However, several other similarly well qualified radiologists who reviewed the same film found no evidence of complicated pneumoconiosis (DX 26, 27, 31, 35, 37, 39, 46 and 54). Further, one subsequent chest x-ray and one subsequent CT scan failed to reveal complicated pneumoconiosis (DX 53). And importantly, as mentioned above, the autopsy and biopsy evaluations did not identify the presence of complicated pneumoconiosis. Accordingly, I find Dr. Mathur's interpretation does not establish the presence of complicated pneumoconiosis.

In summary, although two radiologists identified large opacities in two chest x-rays, other medical evidence, including the particularly probative autopsy evidence, demonstrates that Mr. Parsons did not have complicated pneumoconiosis. As a result, total disability is not established through the irrebuttable presumption under 20 C.F.R. § 718.204 (b).

Pulmonary Function Tests

Exhibit	Date / Doctor	Age / Height	FEV_1 pre[14] post[15]	FVC pre post	MVV pre post	% FEV_1 / FVC pre post	Qualified[16] pre post	Comments
DX 47	3/2/79 Dr. Fleenor	43 67"	3.29	4.31	144	76%	No	
DX 47	4/1/87 Dr. Paranthaman	51 67"	2.83	3.74	127	76%	No	
DX 47	7/8/88 Dr. Miller	52 68"	2.33 2.42	2.81 3.08	88 71	83% 79%	No No	Probably invalid - Dr. Abrahams. Invalid - Dr. Fino (DX 47).
DX 47	1/17/90 Dr. Buddington	54 66.5"	2.55	3.33	96	77%	No	Acceptable - Dr. Zaldivar & Dr. Dahhan. Invalid - Drs. Vest and Morgan (DX 47).
DX 47	4/20/90 Dr. Nash	54 68"	2.17 2.50	2.80 3.35	88.6 110.3		No No	Invalid - Drs. Zaldivar, Renn, Vest, Dahhan & Fino (DX 47).
DX 47	6/18/90 Dr. Sargent	54 66"	2.33 2.55	3.14 3.62	98	74% 70%	No No	
DX 47	4/9/92 Dr. Taylor	56 68"	1.85	2.38	-----	78%	Yes[17]	Invalid - Drs. Michos, Renn, Fino and Morgan (DX 47).

[14]Test result before administration of a bronchodilator.

[15]Test result following administration of a bronchodilator.

[16]Under 20 C.F.R. § 718.204 (b)(2)(i), to qualify for total disability based on pulmonary function tests, for a miner's age and height, the FEV1 must be equal to or less than the value in Appendix B, Table B1 of 20 C.F.R. § 718 (2001), **and either** the FVC has to be equal or less than the value in Table B3, or the MVV has to be equal **or** less than the value in Table B5, or the ratio FEV1/FVC has to be equal to or less than 55%.

[17]The qualifying FEV_1 number is 1.97 for age 56 and 68". The associated qualifying FVC and MVV values are 2.50 & 79.

DX 47	6/22/93 Dr. Sargent	57 66"	1.70 1.90	2.65 2.82	77	64% 68%	No No	
DX 47	2/23/94 Dr. Dahhan	58 (66.5")[18]	1.60 1.72	3.31 2.68	59.65	48% 64%	Yes[19] No	
DX 10, DX 14	9/24/96 Dr. Taylor	61 68"	1.89 1.89	2.56 2.43	66.48 76.98	74% 78%	Yes[20] No	Valid - Dr. Ranavaya (DX 11). Invalid - Dr. Fino, Dr. Castle, and Dr. Morgan (DX 51, DX 52, & DX 54).
DX 26	2/19/97 Dr. Dahhan	61 66.5"	1.93 1.81	2.71 2.41	53.88 46.91	71% 75%	No No	Good patient effort.& understanding. Invalid - Dr. Fino and Dr. Castle (DX 51 & DX 55).
DX 53	9/19/97 Dr. Smiddy	62 68"	2.03	2.37	----	86%	No	Good patient effort and poor understanding. Invalid - Dr. Dahhan and Dr. Castle (DX 55 & DX 52).

Under the provisions of 20 C.F.R. § 718.204 (c) (1), if the preponderance of the pulmonary function tests qualify under Appendix B of Section 718, then in the absence of evidence to the contrary, the pulmonary test evidence shall establish a miner's total disability. To apply this regulatory section requires a five step process. First, an administrative law judge must determine whether the tests conform to the pulmonary function test procedural requirements in 20 C.F.R. § 718.103. Second, the results are compared to the qualifying values for the various tests listed in Appendix B to determine whether the test qualifies. Third, an administrative law judge must evaluate any medical opinion that questions the validity of the test results. Fourth, a determination must be made whether the preponderance of the conforming and valid pulmonary function tests supports a finding of total disability under the regulation. Fifth, if

[18]Dr. Dahhan reported Mr. Parsons' height as 63.8. However, most other physicians measured his height from 66" to 68" and in a subsequent February 1997 exam, Dr. Dahhan measured Mr. Parsons' height as 66.5." Consequently, I will use 66.5" for Dr. Dahhan's test.

[19]The qualifying FEV_1 number is 1.81 for age 58 and 66.5". The associated qualifying FVC and MVV values are 2.50 & 72.

[20]The qualifying FEV_1 number is 1.89 for age 61 and 68." The associated qualifying FVC and MVV values are 2.41 & 76.

the preponderance of conforming tests establishes total disability, an administrative law judge then reviews all the evidence of record and determines whether the record contains "contrary probative evidence." If there is contrary evidence, then it must be given appropriate evidentiary weight and a determination is made to see if it outweighs the pulmonary function tests that support a finding of total respiratory disability. *Fields v. Island Creek Coal Co*., 10 B.L.R. 1-19, 1-21 (1987).

With these principles in mind, I first note that the tests appear to conform to regulatory requirements and 3 of the 12 tests qualify to show total disability under the regulations. Turning to the third step, all of the pulmonary tests generated disputes concerning the validity based on Mr. Parsons' efforts during testing. To determine the probative value of the these tests, I must resolve the dispute among medical experts.

In assessing the reliability of a study, an administrative law judge may give evidentiary weight to the opinion of a physician who reviewed the tracings. *Street v. Consolidation Coal Co*., 7 B.L.R. 1-65 (1984). As the same time, more weight may be given to the observations of a technician who administered the test than to the doctor who reviewed the tracings. *Revnack v. Director, OWCP*, 7 B.L.R. 1-771 (1985). In fact, if a judge credits the expert's opinion over the observations of the person who actually observed the test, he or she must provide a specific rationale. *Brinkley v. Peabody Coal Co*., 14 B.L.R. 1-147 (1990). Little or no weight may be accorded to a pulmonary study which demonstrates "poor" cooperation or comprehension. *Houchin v. Old Ben Coal Co*., 6 B.L.R. 1-1141 (1984). However, if "fair" effort is recorded on the study, the test may be found valid. *Laird v. Freeman United Coal Co*., 6 B.L.R. 1-883 (1984). Finally, even if a non-qualifying test is deficient because a claimant may not have given his or her best effort, the test may still be relevant because it shows that the pulmonary function (even at reduced effort) was still higher than the total disability threshold. *See Anderson v. Youghiogheny & Ohio Coal Co*., 7 B.L.R. 1-152, 1-154 (1984).

Several physicians who revealed the pulmonary studies from July 1988, January 1990, April 1990, April 1992, and September 1997 considered the studies to be invalid. However, despite their concerns, I note that these respective tests, even with purportedly insufficient effort nevertheless did not meet the total disability threshold and thus remain relevant evidence on the absence of totally disabling pulmonary function impairment.

The three experts, Dr. Fino, Dr. Castle, and Dr. Morgan, who reviewed the September 1996 pulmonary function test found it invalid due premature termination of exhalation. On the other hand, the technician who actually observed Mr. Parsons' test found his effort adequate, and Dr. Ranavaya subsequently validated the study. Based principally on the technician's observations, I find this test valid.

Dr. Fino questioned the validity of the February 1997 pulmonary study because Mr. Parsons prematurely terminated his exhalation. Dr. Castle opined the test was technically invalid. The technician who administered the test characterized his effort and cooperation as "good." In addition, Dr. Dahhan, who invalidated the later September 1998 test, decided this earlier test was valid enough to help him with his diagnosis. Again, based on the technician's comments and Dr. Dahhan's use of the test, I find this test also valid.

In summary, all of the pulmonary function studies remain relevant on the issue of total disability. Of the 12 tests, only 3 exceeded the total disability thresholds. As a result, I find the preponderance of the pulmonary function test evidence does not establish total disability under 20 C.F.R. § 718.204 (b) (2) (i).

Arterial Blood Gas Studies

Exhibit	**Date / Doctor**	**$_pCO_2$ (rest)** **$_pCO_2$ (exercise)**	**$_pO_2$ (rest)** **$_pO_2$ (exercise)**	**Qualified**	**Comments**
DX 47	3/29/79 Dr. Paranthaman	35.8 33.1	65 84.7	No[21] No[22]	
DX 47	4/1/87 Dr. Paranthaman	36.5	70.9	No[23]	
DX 47	7/8/88 Dr. Miller	34.2	92	No	
DX 47	1/17/90 Dr. Buddington	31.0 36.1 34.0 35.8	81.3 74.3 81.2 73.6	No[24] No No No	
DX 47	4/20/90 Dr. Nash	39.0 39.0 37.0	136.6 136.6 76.8	No[25] No No	
DX 47	6/18/1990 Dr. Sargent	36.8	69.4	No[26]	
DX 47	8/1/91 Dr. Smiddy	41	83	No[27]	
DX 47	6/22/93 Dr. Sargent	34.1	66.6	No	

[21]For the $_pCO_2$ value of 36, the qualifying $_pO_2$ is 64.

[22]For the $_pCO_2$ value of 33, the qualifying $_pO_2$ is 67.

[23]For the $_pCO_2$ value of 37, the qualifying $_pO_2$ is 63.

[24]For the $_pCO_2$ value of 31, the qualifying $_pO_2$ is 69.

[25]For the $_pCO_2$ value of 39, the qualifying $_pO_2$ is 61.

[26]For the $_pCO_2$ value of 37, the qualifying $_pO_2$ is 63.

[27]For the $_pCO_2$ values of 40-49, the qualifying $_pO_2$ is 60.

DX 47	2/23/94 Dr. Dahhan	35.0	71.5	No[28]	Minimum hypoxia.
DX 14, DX 16	9/23/96 Dr. Taylor	35.6 34.6	71 81	No No	
DX 26	2/19/97 Dr. Dahhan	36.5	70.5	No	Minimum hypoxia.

Since none of the arterial blood gas studies meet the regulatory standards for total disability, total disability is not established under 20 C.F.R. § 718.204 (b) (2) (ii).

Medical Opinion

When total disability cannot be establish based on the presence of cor pulmonale, complicated pneumoconiosis, pulmonary function tests, or arterial blood gas studies, a claimant may still establish total disability through reasoned medical opinion. According to 20 C.F.R. § 718.204 (b) (2) (iv), total disability may be found:

> if a physician exercising reasoned medical judgment, based on medically acceptable clinical and laboratory diagnostic techniques, concludes that a miner's respiratory or pulmonary condition prevents or prevented the miner from engaging in employment as described in paragraph (b) of this section.

The regulation, 20 C.F.R. § 718.204 (b) (1) defines such employment as either his usual coal mine work or other gainful employment requiring comparable skills. Thus, to evaluate total disability under these provisions, an administrative law judge must compare the exertional requirements of the claimant's usual coal mine employment with a physician's assessment of his respiratory impairment. *Schetroma v. Director, OWCP*, 18 B.L.R. 1-19 (1993).

Based on his work histories and hearing testimony, I find Mr. Parsons engaged in moderate manual labor. Although he spent most of his time in a seated position operating the shuttle car, he had to load and unload supplies and equipment which required an ability to lift at least twenty pounds and carry blocks at least twenty feet (DX 26 - reference by Dr. Dahhan, DX 47 - 1990 hearing, page 21; 1994 hearing, page 12; and, 1985 Form CM 913).

Prior to considering the dozens of medical assessments and opinions in this case, I will summarize the massive radiographic evidence. Although the parties have stipulated to the presence of pneumoconiosis, and the pathologic evidence clearly indicates Mr. Parsons had simple coal workers' pneumoconiosis, a review of the radiographic record will facilitate an understanding of some of the physicians' opinions. I will also add that although many physicians found evidence of pneumoconiosis in the chest x-rays, the majority of the interpretations, including the preponderance of the opinions of the better qualified doctors who

[28]For the $_{P}CO_2$ value of 35, the qualifying $_{P}O_2$ is 65.

were board certified radiologists and B readers, found the radiographic evidence to be negative for the presence of pneumoconiosis.

Chest X-Rays

Date of x-ray	**Exhibit**	**Physician**	**Interpretation**
August 7, 1968	DX 47	Setzler	(Negative for pneumoconiosis)[29] There appears to be a slight patchy infiltration extending beyond the hilar margins, especially on the right.
September 1, 1971	DX 47	Taylor	(Inconclusive for pneumoconiosis)[30] Minimal fibrosis bilaterally.
December 3, 1971	DX 47	JWP	Completely negative.
(same)	DX 47	JLB	Negative for pneumoconiosis.
May 8, 1973	DX 47	Kinser	(Inconclusive for pneumoconiosis) Minimal fibrosis bilaterally
April 10, 1979	DX 47	Saba, B, BCR[31]	Completely negative.
(same)	DX 47	Scott, B, BCR	Completely negative.
(same)	DX 47	Wheeler, B, BCR	Completely negative.
(same)	DX 32 and 47	Fleenor	Positive for pneumoconiosis category 1/1,[32] type p opacities.[33]

[29]Since a physician evaluating a chest x-ray can be expected to accurately report the presence of any abnormalities, an administrative law judge may infer that the absence of a mention of pneumoconiosis indicates pneumoconiosis was not present. *See Marra v. Consolidation Coal Co.* 7 BLR 1-216, 1-219 (1985).

[30]Although Dr. Taylor identified pulmonary fibrosis, he neither diagnosed pneumoconiosis nor provided profusion and opacity observations supportive of a finding of pneumoconiosis.

[31]The following designations apply: C- C reader, B – B reader, and BCR – Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A "C Reader" designates only highly regarded individuals who developed the black lung classification system for chest x-rays and represents the highest interpreter qualification A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and examination, as proficient in interpreting x-ray films of all kinds including images of the lungs.

[32]The profusion (quantity) of the opacities (opaque spots) throughout the lungs is measured by four categories: 0 = small opacities are absent or so few they do not reach a category 1; 1 = small opacities definitely present but few in number; 2 = small opacities numerous but normal lung markings are still visible; and, 3 = small opacities very numerous and normal lung markings are usually partly or totally obscured. An interpretation of category 1, 2, or 3 means there are opacities in the lung which may be used as evidence of pneumoconiosis. If the interpretation is 0, then the assessment is not evidence of pneumoconiosis. A physician will usually list the interpretation with two digits. The first digit is the final assessment; the second digit represents the category that the doctor also seriously considered. For example, a reading of 1 / 2 means the doctor's final determination is category 1 opacities but he considered placing the interpretation in category 2. Or, a reading of 0/0 means the doctor found no, or few, opacities and didn't see any marks that would cause him or her to seriously consider category 1. According to 20 C.F.R. § 718.102 (b) (2001), a profusion of 0/1 does not constitute evidence of pneumoconiosis.

(same)	DX 32 and 47	Ramakrishnan, BCR	Positive for pneumoconiosis category 1/0, type q opacities.
April 19, 1979	DX 32	Fleenor	Positive for pneumoconiosis, category 1/1, type p opacities.
(same)	DX 32	Ramakrishnan, BCR	Positive for pneumoconiosis, category 1/0, type q opacities.
August 3, 1979	DX 25, DX 27	Cunningham, B, BCR	Negative for pneumoconiosis. The pulmonary markings are somewhat heavy as from mild congestion and perhaps a little non-specific scarring.
(same)	DX 25, DX 27, 135	Malloy	Negative for pneumoconiosis. Classification would be 0 and at most 0/1.
(same)	DX 135	Cooper	Negative for pneumoconiosis.
August 4, 1979	DX 32	Straughan, B, BCR	Positive for pneumoconiosis, category 1/1, type p opacities. Lungs are otherwise essentially clear.
August 21, 1982	DX 25, 27, 136	Felson, C, B, BCR	Completely negative.
(same)	DX 25, 27, 135	Wiot, B, BCR	Completely negative.
(same)	DX 25, 27, 135	Pendergrass, B, BCR	Negative for pneumoconiosis.
(same)	DX 25,27	Bassham	Negative for pneumoconiosis.
(same)	DX 27	Cunningham, B, BCR	Negative for pneumoconiosis.
(same)	DX 27	Morgan, B	Negative for pneumoconiosis.
(same)	DX 27	Wheeler, B, BCR	Completely negative.
(same)	DX 27	Spitz, B, BCR	Completely negative.
(same)	DX 27	Gale, B, BCR	Negative for pneumoconiosis. The classification is category 0/1, q/p.
(same)	DX 32	White, BCR	Positive for pneumoconiosis. The ILO classification is 1/1, type s opacities.
(same)	DX 32	Patel, BCR	Positive for pneumoconiosis, category 2/1, type p

[33]There are two general categories of small opacities defined by their shape: rounded and irregular. Within those categories the opacities are further defined by size. The round opacities are: type p (less than 1.5 millimeter (mm) in diameter), type q (1.5 to 3.0 mm), and type r (3.0 to 10.0 mm). The irregular opacities are: type s (less than 1.5 mm), type t (1.5 to 3.0 mm) and type u (3.0 to 10.0 mm). JOHN CRAFTON & ANDREW DOUGLAS, RESPIRATORY DISEASES 581 (3d ed. 1981).

			opacities.
(same)	DX 32	Ramakrishnan, BCR	Positive for pneumoconiosis, category 1/0, type q opacities .
(same)	DX 32	Bassali, B, BCR	Positive for pneumoconiosis, category 1/1, type q/t opacities.
(same)	DX 32	Brandon, B, BCR	Positive for pneumoconiosis, category 1/1, type p opacities.
(same)	DX 32	Penman	Positive for pneumoconiosis, category 1/1, type p opacities.
May 8, 1985	DX 47	Buonocore	(Negative for pneumoconiosis) Left hemidiaphragm and dense calcification of lymph nodes in the left perihilar area. Lungs remain clear and well expanded.
August 25, 1985	DX 47	Wooten, BCR	(Negative for pneumoconiosis). Normal chest.
September 1, 1985	DX 47	Wooten, BCR	(Negative for pneumoconiosis). Normal chest.
September 2, 1985	DX 47	Wooten, BCR	(Negative for pneumoconiosis). Normal chest.
September 30, 1986	DX 47	Navani, BCR, A[34]	(Negative for pneumoconiosis). The lung fields are free of active pneumonic infiltrative process. Slight prominence of the left hilum.
(same)	DX 54, DX 140	Morgan, B	Negative for pneumoconiosis, category 0/0. Possible pneumonia.
(same)	DX 47	Sargent [J. Dale], B	Negative for pneumoconiosis.
(same)	DX 141	Dahhan, A[35]	Negative for pneumoconiosis.
February 6, 1987	DX 47	Navani, BCR, A	(Inconclusive for pneumoconiosis). Accentuated lung markings.
(same)	DX 103	Lippmann, B	Completely negative.
April 1, 1987	DX 47	Green, B	Negative for pneumoconiosis.
(same)	DX 47	Navani, BCR, A	Negative for pneumoconiosis. Profusion category 0/1, type s/s opacities.
(same)	DX 47	Saba, B, BCR	Completely negative.
(same)	DX 47	Scott, B, BCR	Completely negative.
(same)	DX 47	Wheeler, B, BCR	Completely negative.

[34]From 1/1/86 through 4/30/87.

[35]From 8/1/91 through 11/30/95, date of interpretation 7/18/94.

October 6, 1987	DX 47	Navani, B, BCR	(Negative for pneumoconiosis) Slight increased lung markings are noted. No evidence of pneumonic consolidation or any significant cardiopulmonary disease process is identified.
(same)	DX 104	Lippmann, B	Completely negative.
July 8, 1988	DX 47	Spitz, B, BCR	Completely negative.
(same)	DX 47	Shipley, B, BCR	Negative for pneumoconiosis.
(same)	DX 47	Wiot, B, BCR	Completely negative.
October 5, 1988	DX 47	Saha	(Negative for pneumoconiosis). The lung fields are free of active pneumonic infiltrative process.
(same)	DX 47	Spitz, B, BCR	Completely negative.
(same)	DX 47	Wiot, B, BCR	Completely negative.
(same)	DX 47	Shipley, B, BCR	Completely negative.
(same)	DX 47, DX 141	Dahhan, A	Completely negative.
(same)	DX 54, DX 140	Morgan, B	Negative for pneumoconiosis, category 0/0.
(same)	DX 47	Sargent [J. Dale], B	Negative for pneumoconiosis.
January 5, 1990	DX 47	Sargent [E.N.], B, BCR	Negative for pneumoconiosis.
(same)	DX 47	Saha	(Inconclusive for pneumoconiosis). Linear densities are noted at the left lower lung field which is probably chronic in nature. Any active inflammatory process or any other abnormality cannot be excluded entirely. There is pleural thickening in the left lateral chest.
January 15, 1990	DX 47	Sargent [E.N.], B, BCR	Negative for pneumoconiosis.
(same)	DX 47	Navani, B, BCR	(Negative for pneumoconiosis). Pleural thickening along the left lateral chest wall is seen.
April 20, 1990	DX 47	Nash	Positive for pneumoconiosis, category 2/1, type p/p opacities.
(same)	DX 47	Spitz, B, BCR	Negative for pneumoconiosis.
(same)	DX 47	Shipley, B, BCR	Negative for pneumoconiosis.
(same)	DX 47, DX 141	Dahhan, A	Negative for pneumoconiosis.
(same)	DX 47	Wiot, B, BCR	Negative for pneumoconiosis.

(same)	DX 54, DX 140	Morgan, B	Negative for pneumoconiosis, category 0/0. Prominent left hilum.
(same)	DX 47	Sargent [J. Dale], B	(Inconclusive for pneumoconiosis). Linear irregular densities. Although the ILO 1980 standard system requires classification, these irregular opacities are not caused by coal workers' pneumoconiosis [deposition testimony].
April 21, 1990	DX 47	Ramakrishnan, BCR	Positive for pneumoconiosis, category 1/1, type p/s opacities.
(same)	DX 47	Abramowitz, B, BCR	Negative for pneumoconiosis, category 0/1, type s/t opacities.
(same)	DX 47	Gogineni, B, BCR	Negative for pneumoconiosis, category 0/1, type s/t opacities.
June 18, 1990	DX 47	Sargent, B	Positive for pneumoconiosis, category 1/1, type t/t opacities.
(same)	DX 47	Spitz, B, BCR	Completely negative.
(same)	DX 47	Wiot, B, BCR	Negative for pneumoconiosis.
(same)	DX 47	Shipley, B, BCR	Completely negative.
(same)	DX 54	Morgan, B	Negative for pneumoconiosis, category 0/0.
February 1, 1991	DX 47	Sargent, B, BCR	Negative for pneumoconiosis.
(same)	DX 47	Saha	(Inconclusive for pneumoconiosis). Chronic pulmonary disease. No acute process.
July 30, 1991	DX 80	Phillips	(Inconclusive for pneumoconiosis) Linear stranding opacities.
August 1, 1991	DX 47	Smiddy	(Negative for pneumoconiosis) [serial chest x-rays] Minimal effusion atelectasis in left lung base.
(same)	DX 80	Sinder	(Inconclusive for pneumoconiosis) Pulmonary interstitial markings, mildly diffuse.
(same)	DX 80	McMurray	(Inconclusive for pneumoconiosis) Moderate interstitial markings.
August 3, 1991	DX 80	McMurray	(Inconclusive for pneumoconiosis) Moderate interstitial congestion.
August 4, 1991	DX 80	Cooper	(Negative for pneumoconiosis). No acute lung disease.
October 23, 1992	DX 47	Spitz, B, BCR	Completely negative.
(same)	DX 47,	Dahhan. A	Negative for pneumoconiosis category 0/1, type

	DX 141		s/s opacities.
(same)	DX 54, DX 140	Morgan, B	Negative for pneumoconiosis, category 0/0. Linear atelectasis left mid zone.
April 16, 1993	DX 105	Lippmann, B	Negative for pneumoconiosis. Pulmonary edema present.
May 14, 1993	DX 106	Lippmann, B	Negative for pneumoconiosis.
June 22, 1993	DX 47	Spitz, B, BCR	Negative for pneumoconiosis.
(same)	DX 47	Shipley, B, BCR	Negative for pneumoconiosis. Focal scarring left mid zone and left lower zone.
(same)	DX 47	Wiot, B, BCR	Negative for pneumoconiosis. Fibrotic change left mid lung.
(same)	DX 47	Sargent, B	Positive for pneumoconiosis, category 1/1, type t/t opacities.
February 23, 1994	DX 47	Dahhan, A	Negative for pneumoconiosis, category 0/1, type s/t opacities.
(same)	DX 47	Spitz, B, BCR	Negative for pneumoconiosis. Infiltrate and/or nodule in left mid lung.
(same)	DX 47	Shipley, B, BCR	Negative for pneumoconiosis. Focal scarring left mid zone and left lower zone.
(same)	DX 54, DX 140	Morgan, B	Negative for pneumoconiosis, category 0/0. Pleural thickening - left. Linear atelectasis left mid zone and left lower zone. Linear markings left lower zone.
January 31, 1995	DX 107	Lippmann, B	Negative for pneumoconiosis.
March 6, 1995	DX 108	Lippmann, B	Negative for pneumoconiosis.
July 14, 1995	DX 109	Lippmann, B	Negative for pneumoconiosis. Pulmonary edema present.
December 6, 1995	DX 110	Lippmann, B	Negative for pneumoconiosis.
January 15, 1996	DX 111	Lippmann, B	Negative for pneumoconiosis.
April 15, 1996	DX 100	Saha	(Negative for pneumoconiosis). Improvement in pneumonia.
April 24, 1996	DX 27	Taylor	(Negative for pneumoconiosis) No sign of acute inflammatory process although there is some scar tissue and some evidence of previous pneumonia in the left lung.
(same)	DX 112	Lippmann, B	Negative for pneumoconiosis.
September 23, 1996	DX 17	Lippmann, B	Positive for pneumoconiosis, category 1/1, type s/t opacities.

(same)	DX 18, DX 100	Lee	(Inconclusive for pneumoconiosis). Peripheral right mid lung demonstrates a 1 cm density. Peripheral left lower lung demonstrates increased interstitial and bronchovascular marking present previously and significant change is not seen. Findings may represent scar. Remainder of lung fields are clear.
(same)	DX 31	Wheeler, B, BCR	Negative for pneumoconiosis. 1.5 x 2 cm mass in periphery left mid lung involving pleura... with few scars in adjacent lung due to healed inflammatory disease most likely TB [tuberculosis]. Focal pleural fibrosis on chest walls with few adjacent scars in left mid lung periphery and one in right lung periphery. Several small pleural nodules are due to granulomatous disease. Possible few tiny nodules in portion LUL but no symmetrical pattern to indicate pneumoconiosis.
(same)	DX 37	Fishman, B, BCR	Negative for pneumoconiosis. The patient has several areas of fine pleural thickening in the left chest. There is a fine focal pleural thickening in the right chest. Minimal scarring in the anterior portion of the right upper lung. Ill defined nodule or inflammatory mass present in the left upper lung. A similar change but to a lesser degree is present in the right chest with some associated parenchymal disease near an area of pleural thickening. No other abnormalities noted.
(same)	DX 40	Fino, B	Negative for pneumoconiosis. No changes consistent with a coal mine dust associated occupational lung disease.
October 27, 1996	DX 27, DX 100	Taylor	(Negative for pneumoconiosis) COPD (chronic obstructive pulmonary disease) present. No active infiltrate. No pleural effusion.
November 27, 1996	DX 113	Lippmann, B	Negative for pneumoconiosis. Pulmonary edema present.
February 19, 1997	DX 26	Dahhan, B	Negative for pneumoconiosis.
(same)	DX 27, DX 143	Wiot, B, BCR	Negative for pneumoconiosis. There is old pleural disease on the left, with some stranding from the left hilum towards this pleural disease, consistent with post-operative change. In addition, in the right fourth anterior interspace, there is a poorly defined approximately 1 cm nodule which must be considered as a malignancy until proven

			otherwise. It is not related to coal dust exposure.
(same)	DX 31, DX 143	Spitz, B, BCR	Negative for pneumoconiosis. Module right mid lung. Infiltrate and stranding in left mid lung.
(same)	DX 35, DX 143	Shipley, B, BCR	Negative for pneumoconiosis. Right sided pulmonary nodule. Left sided focal infiltrate, possibly pneumonia.
(same)	DX 37	Scott, B, BCR	Negative for pneumoconiosis. 1 cm density lateral right mid lung - possible calcified granuloma or scar.
(same)	DX 37	Wheeler, B, BCR	Negative for pneumoconiosis. Probable small calcified granuloma in lateral portion right mid lung or pleura.
(same)	DX 39	Kim, B, BCR	Negative for pneumoconiosis. A focal irregular density in the right mid lung laterally, probable granuloma but need follow-up. Increased densities in the left mid chest parenchyma fibrosis or infiltrate.
(same)	DX 43 and 46	Mathur, B, BCR	Positive for pneumoconiosis, category 2/3, type q/t opacities. Large type A opacity. Pleural thickening consistent with pneumoconiosis, right chest wall width A, extent 2 and left chest wall, width A, extent 2.
(same)	DX 46	Pathak, B	Positive for pneumoconiosis, category 2/2, type p/q opacities. Bilateral pleural thickening consistent with pneumoconiosis. Ill defined nodule periphery of right mid zone which may represent a poorly calcified granuloma or a small soft tissue density parenchymal nodule.
(same)	DX 46	Cappiello, B, BCR	Positive for pneumoconiosis, category 2/3, type p/q opacities. Indeterminate nodule right mid lung zone.
(same)	DX 54	Morgan, B	Negative for pneumoconiosis, category 0/0. Increased markings left lower zone.
December 10, 1997	DX 53	Smiddy	(Negative for pneumoconiosis) Five-lobe scarring with old focal scaring left hilum and left chest laterally. No active process.

CT Scans

October 22, 1996	DX 14, DX 19, DX 100	Lee	Abnormal chest. Posterior peripheral right midlung zone area demonstrates a focal linear increased attenuation, which may represent scarring. The peripheral anterior left lower lung, however, demonstrates lobulated soft tissue

			nodular density which may represent a pleural-based mass.
(same)	DX 142	Wheeler, BCR, B	1.5 x 2. cm mass, most likely healed inflammatory disease.
(same)	DX 142	Fino, B	Negative for pneumoconiosis.
(same)	DX 142	Fishman, BCR	Ill defined nodule; no evidence of pneumoconiosis or silicosis.
September 15, 1997 [CT Scan]	DX 53	Phillips	Specific pathologic soft tissue densities involving the pulmonary pleuroparenchymal margins in the lateral mid portions of each lung. These are non-calcified with considerations including pulmonary fibrosis, scarring and neoplasm.

Dr. L. J. Fleenor

On April 10, 1979, Dr. Fleenor, board certified in family medicine,[36] performed a complete physical examination, chest x-ray, pulmonary function study and arterial blood gas study. The physician recorded a coal mine employment history of twenty-three years and a cigarette smoking history of twenty pack years.[37] At the physical examination, Mr. Parsons offered subjective complaints of cough, phlegm, dyspnea, orthopnea and edema. The physician interpreted Mr. Parsons' chest x-ray as positive for pneumoconiosis, category 1/1, type p opacities. The pulmonary function study and arterial blood gas study produced non-qualifying values. In the final analysis, Dr. Fleenor diagnosed black lung arising from coal dust exposure. At the same time, according to Dr. Fleenor, Mr. Parsons could still work in the coal mines, but could no longer play softball.

Dr. S. K. Paranthaman
(DX 47)

On April 1, 1987, Dr. Paranthaman, board certified in pulmonary disease and internal medicine,[38] performed a complete physical examination, chest x-ray, pulmonary function study and arterial blood gas study. The physician recorded a coal mine employment history of twenty-eight years and a cigarette smoking history of two packs of cigarettes per day for thirty-two years with present consumption at one and a half packs of cigarettes per day. Mr. Parsons complained about cough, sputum, dyspnea, chest pain and nocturnal dyspnea. The chest examination was essentially normal. A radiologist interpreted Mr. Parsons' chest x-ray as negative for pneumoconiosis, category 0/1. The pulmonary function study and arterial blood gas study produced non-qualifying values. In the final analysis, Dr. Paranthaman diagnosed chronic

[36]As I advised the parties at the hearing (TR, page 6-7), I take judicial notice of Dr. Fleenor's board certified and have attached the certification documentation.

[37]A pack year equals the consumption of one pack of cigarette per day for one day.

[38]I take judicial notice of Dr. Paranthaman's board certification and have attached the certification documentation.

bronchitis caused by cigarette smoking, although he conceded that coal dust exposure could have aggravated this pulmonary condition.

On May 18, 1988, Dr. Paranthaman provided an additional medical opinion. After reconsidering Mr. Parsons' employment history, physical examination and laboratory test results, Dr. Paranthaman concluded that Mr. Parsons suffers from chronic bronchitis without significant airflow obstruction. This chronic bronchitis causes only mild functional impairment and therefore, from a respiratory perspective, Mr. Parsons could return to the coal mines. However, Dr. Paranthaman opined that Mr. Parsons' back injury would prevent any physical labor.

Dr. J. D. Miller
(DX 47)

On July 8, 1988, Dr. Miller performed a complete physical examination, chest x-ray, pulmonary function study and arterial blood gas study. The physician recorded a coal mine employment history of thirty-two years and a cigarette smoking history of one and a half to two packs of cigarettes per day for thirty years. Mr. Parsons described dyspnea, orthopnea, cough, sputum and wheezing. The chest examination revealed a chest clear to percussion and auscultation. The chest x-ray revealed diffuse fibronodular infiltrate of 1/1 profusion. The pulmonary function study and arterial blood gas study produced non-qualifying values for total disability. Based on the chest x-ray, Dr. Miller stated, "This patient does have chest x-ray changes of pneumoconiosis." The physician did not provide any comment on Mr. Parsons' pulmonary impairment.

Dr. Roger A. Abrahams
(DX 47)

On February 26, 1990, Dr. Abrahams, board certified in pulmonary disease and internal medicine, provided a reasoned medical opinion after considering Mr. Parsons' employment history, smoking history, twelve interpretations of four chest x-rays, four pulmonary function studies and four arterial blood gas studies. In the final analysis, Dr. Abrahams stated that Mr. Parsons does not suffer from coal workers' pneumoconiosis or any respiratory disability. However, the retired coal miner does suffer from minimal resting hypoxia caused by chronic bronchitis due to Mr. Parsons' lengthy cigarette smoking history. According to Dr. Abrahams, the official record does not contain sufficient information to offer a medical opinion concerning Mr. Parsons' total disability from cervical disc syndrome.

Dr. Arthur J. Nash
(DX 47)

On April 20, 1990, Dr. Nash, board certified in anesthesiology, performed a complete physical examination, chest x-ray, pulmonary function study and arterial blood gas study. The physician recorded a coal mine employment history of thirty-three years, with Mr. Parsons retiring in May 1985 after a coal mine accident. In addition, Dr. Nash recorded a cigarette smoking history of thirty-five years at half to one and a half packs of cigarettes per day. The retired coal miner offered subjective complaints of dyspnea, chronic productive cough, sputum

and orthopnea. The chest examination revealed moderate dyspnea. The physician interpreted Mr. Parsons' chest x-ray as positive for pneumoconiosis, category 2/1, type p/p opacities. The pulmonary function study and arterial blood gas study did not establish total disability. After considering Mr. Parsons' employment history, smoking history, physical examination and objective medical information, Dr. Nash concluded that Mr. Parsons suffers from chronic bronchitis, chronic obstructive lung disease and coal workers' pneumoconiosis. According to the physician, Mr. Parsons is totally and permanently disabled "on the basis of his pulmonary problems alone," with a majority of this respiratory impairment attributable to Mr. Parsons' coal mine employment.

Dr. J. Dale Sargent
(DX 47)

On June 18, 1990, Dr. Sargent, board certified in pulmonary disease, internal medicine, and critical care, performed a complete physical examination, chest x-ray, pulmonary function study and arterial blood gas study. The physician recorded a coal mine employment history of thirty-three years and a cigarette smoking history of thirty years at one and a half packs of cigarettes per day. At the physical examination, Mr. Parsons complained about dyspnea, cough, sputum, hemoptysis, wheezes, chest pain and orthopnea. The chest examination revealed faint expiratory wheezing and slightly decreased breath sounds. The physician interpreted Mr. Parsons' chest x-ray as positive for pneumoconiosis, category 1/1, type t/t opacities. The pulmonary function study and arterial blood gas study produced non-qualifying values. In the final analysis, Dr. Sargent diagnosed coal workers' pneumoconiosis. However, according to Dr. Sargent, the retired coal miner did not suffer from any restrictive ventilatory impairment, and only minimal obstructive ventilatory impairment. The physician believed Mr. Parsons' minimal obstructive impairment was caused by cigarette smoking. In any event, Dr. Sargent stated that Mr. Parsons respiratory impairment would not preclude a return to coal mine employment, although the cervical disc syndrome would prevent Mr. Parsons from operating a shuttle car in the coal mines.

In a deposition conducted on August 27, 1990, Dr. Sargent corrected Mr. Parsons' smoking history to two packs of cigarettes per day for thirty years. In addition, Dr. Sargent stated that Mr. Parsons' chest x-ray film, profusion 1/1, type "t" opacities in the lower zones is not a normal pattern for coal workers' pneumoconiosis. In general, a chest x-ray which is positive for coal workers' pneumoconiosis contains rounded opacities (type p, q or r opacities) in the upper zones. Therefore, after repeated questioning, Dr. Sargent conceded that Mr. Parsons "may or may not have simple coal workers' pneumoconiosis."

On June 22, 1993, Dr. Sargent performed a second physical examination, chest x-ray, pulmonary function study and arterial blood gas study. The physician recorded the same coal mine employment and cigarette smoking histories. However, after suffering a heart attack, Mr. Parsons had reduced his cigarette smoking to only three cigarettes per day. At the physical examination, Mr. Parsons complained about dyspnea, chest pain, chronic productive cough, wheezing, orthopnea and edema. The chest examination revealed clear lungs sounds with no wheezing or crackles. Dr. Sargent interpreted Mr. Parsons' chest x-ray as positive for pneumoconiosis, category 1/1, type t/t opacities. The pre-bronchodilator pulmonary function

study produced a qualifying value showing total disability, which according to Dr. Sargent indicated moderate obstructive impairment, with no restrictive impairment. The post-bronchodilator pulmonary function study and arterial blood gas study produced non-qualifying values. In conclusion, Dr. Sargent stated that Mr. Parsons "may be suffering from simple coal workers' pneumoconiosis" based on a positive chest x-ray interpretation. Once again, however, Dr. Sargent stated that coal workers' pneumoconiosis rarely produces type "t" opacities in the lower lung zones. According to the physician, Mr. Parsons suffers from a sufficient respiratory impairment to prevent a return to coal mine employment. As Mr. Parsons did not suffer from any significant respiratory impairment in a June 1990 physical examination, Dr. Sargent attributed Mr. Parsons' respiratory impairment to chronic cigarette smoking and his thoracic cage abnormalities following coronary artery bypass surgery. The physician excluded coal dust exposure as a possible cause of Mr. Parsons' respiratory impairment by observing that coal workers' pneumoconiosis causes a mixed restrictive and obstructive ventilatory impairment, and Mr. Parsons' pulmonary function values do not reveal any restrictive impairment.

After considering several additional chest x-ray films before a July 14, 1994 deposition, Dr. Sargent once again maintained that Mr. Parsons' irregular opacities in the lower lung zones are not caused by coal dust exposure. According to Dr. Sargent, Mr. Parsons suffers from a moderate respiratory impairment, caused by cigarette smoking. However, Dr. Sargent believed that Mr. Parsons' respiratory impairment would prevent a return to coal mine employment.

Dr. Kelly D. Taylor
(DX 13, DX 15, DX 47, and CX 1)

In several progress notes from March 1988 through January 1990, Dr. Taylor discusses Mr. Parsons' neck pain and blood sugar. In one progress note, Dr. Taylor prescribed medication for acute pleuropneumonitis. On November 6, 1989, Mr. Parsons presented to Dr. Taylor's office complaining of severe pain in the left chest, left arm and left side of the neck. A pulmonary examination revealed occasional rhonchi, but no rales. After several days at Lee County Community Hospital, Dr. Taylor discharged Mr. Parsons with a diagnosis of cervical disc syndrome, chronic bronchitis and diabetes mellitus.

On August 7, 1991, Dr. Taylor opined Mr. Parsons suffers from coal workers' pneumoconiosis based on subjective complaints, chest x-ray evidence and arterial blood gas values. However, the physician conceded that Mr. Parsons' cigarette smoking history may cause some of the chronic fibrotic changes on Mr. Parsons' chest x-rays.

On March 1, 1995, Dr. Taylor admitted Mr. Parsons for two days of hospital observation associated with chest pain. Recently, due to a significant episode of respiratory failure, Mr. Parsons had been intubated and violently struggled in reaction to the treatment. After the struggle, Mr. Parsons reported chest pain. Although Dr. Taylor believed the cause was musculoskeletal, he admitted Mr. Parsons for cardiac assessment. Mr. Parsons' medical history included a "massive" myocardial infarction, subsequent coronary artery by-pass surgery "with complications of Staph infection of chest wall. . . major chest wall surgery with sternectomy and rib resections." Previously, Mr. Parsons had been an extremely heavy cigarette smoker. Upon physical examination, Dr. Taylor heard moderate rales. The physician found no evidence of

congestive heart failure. Dr. Taylor diagnosed non-cardiac chest pain, improved acute respiratory failure, and COPD with pneumoconiosis.

In a letter, dated September 23, 1996, Dr. Taylor, who treated Mr. Parsons for several years, states that Mr. Parsons suffers from significant chronic lung disease which produces bronchospasms, dyspnea and productive cough. According to Dr. Taylor, twenty years of coal mine employment contributed to Mr. Parsons' pulmonary impairment. After completing this letter, Dr. Taylor performed a complete physical examination, chest x-ray, pulmonary function study and arterial blood gas study in January 1997. Mr. Parsons' medical history included arteriosclerotic heart disease, myocardial infarction with subsequent coronary artery by-pass surgery, diabetes, and ruptured neck and back discs with subsequent surgery. At the physical examination, Mr. Parsons offered subjective complaints of sputum, wheezing, dyspnea, cough, chest pain and orthopnea. The physician recorded a one pack per day cigarette smoking habit. The chest examination revealed rhonchi, bronchospasms and basilar rales along with significant instability of the chest wall caused by a sternectomy and prior infection. The pre-bronchodilator pulmonary function study produced values that showed total disability. The post-bronchodilator pulmonary function study did not reach the total disability threshold but did indicate a moderate restrictive lung disease. The arterial blood gas study did not show total disability. In the final analysis, Dr. Taylor diagnosed chronic respiratory insufficiency with congestive heart failure complicated by pneumoconiosis. According to Dr. Taylor, Mr. Parsons' total respiratory impairment prevented a return to coal mine employment or any other work. His pulmonary condition alone was totally disabling considering the pulmonary function tests and x-rays.

Lee County Community Hospital
(DX 47)

On October 15, 1993, Mr. Parsons arrived at Lee County Community Hospital complaining of chest pains. A physician recorded a lengthy coal mine employment history and cigarette smoking history along with a long history of heart artery disease. A chest examination revealed mild scattered rhonchi bilaterally. The physician admitted Mr. Parsons to the critical care unit to observe the retired coal miner's cardiac status.

Dr. A. Dahhan
(DX 26, DX 47, DX 138, DX 141, DX 147, DX 81, and EX 3)

On February 23, 1994, Dr. Dahhan, board certified in pulmonary disease and internal medicine, performed a complete physical examination, chest x-ray, pulmonary function study and arterial blood gas study. The physician recorded a coal mine employment history of thirty-three years and a cigarette smoking history of forty years at half to one pack of cigarettes per day. At the physical examination, Mr. Parsons offered subjective complaints of cough, sputum, wheezing, dyspnea, chest pain, orthopnea and occasional edema. The chest examination revealed reduced air entry to the lung bases with no wheezing. In addition, the physical examination revealed slight edema. The physician interpreted Mr. Parsons' chest x-ray as negative for pneumoconiosis, category 0/1, type s/t opacities. The pulmonary function study produced non-qualifying values, although Dr. Dahhan indicated that Mr. Parsons failed to cooperate, and therefore the pulmonary function study was invalid. The arterial blood gas study

produced non-qualifying values. After considering Mr. Parsons' employment history and medical records from the official record, Dr. Dahhan concluded that Mr. Parsons does not suffer from coal workers' pneumoconiosis. According to Dr. Dahhan, a majority of the radiologists interpreted Mr. Parsons' chest x-rays as negative for pneumoconiosis, Mr. Parsons' physical examination failed to reveal any crackles, and the pulmonary function studies failed to reveal any indication of a restrictive ventilatory defect. Instead, the physician diagnosed chronic bronchitis caused by a lengthy cigarette smoking history. From a respiratory perspective, Dr. Dahhan believed that Mr. Parsons could return to coal mine employment. However, multiple medical problems including obesity, coronary artery disease, sternum resection, diabetes mellitus, and multiple disc surgery would prevent Mr. Parsons from returning to coal mine employment.

In a deposition conducted on July 20, 1994, Dr. Dahhan considered four additional chest x-ray films, which the physician interpreted as negative for coal workers' pneumoconiosis. After considering these chest x-rays, Dr. Dahhan once again stated that Mr. Parsons does not suffer from coal workers' pneumoconiosis or any totally disabling respiratory impairment.

On February 19, 1997, Dr. Dahhan performed a complete physical examination, chest x-ray, pulmonary function study and arterial blood gas study. The physician recorded an employment history of thirty-three years of coal mine employment and a cigarette smoking history of forty-three years at one-half pack of cigarettes per day. At the physical examination, Mr. Parsons offered subjective complaints of chronic cough, wheezing, exertional dyspnea, orthopnea and edema. The chest examination revealed reduced air entry to both lungs with scattered expiratory wheeze and a prolonged expiratory phase. The medical history included right side weakness since a stroke in 1996 and removal of the sternum due to infection. The pulmonary function study indicated a moderate obstructive pulmonary defect and the arterial blood gas study showed sufficient ventilation. After considering additional medical evidence, including Mr. Parsons' pulmonary function study and arterial blood gas study from September 24, 1996 and a patient questionnaire describing Mr. Parsons' physical requirements as a shuttle car operator, Dr. Dahhan concluded that Mr. Parsons suffers from chronic obstructive lung disease caused by cigarette smoking, not coal dust exposure. According to the physician, Mr. Billy Parsons does not retain sufficient respiratory capacity to operate a shuttle car or any comparative physical employment. Based on his interpretation of an x-ray, he did not believe Mr. Parsons had pneumoconiosis. And, even if Mr. Parsons had pneumoconiosis, Dr. Dahhan did not believe the moderate obstructive defect would be due to simple pneumoconiosis.

In a deposition conducted on March 9, 1998, Dr. Dahhan considered an additional medical report and a pulmonary function study performed by Dr. Smiddy along with comprehensive medical reports from Drs. Fino and Castle (DX 55). Referring to his 1997 examination of Mr. Parsons, Dr. Dahhan noted that the EKG indicated several heart ailments but did not support a finding of cor pulmonale. According to Dr. Dahhan, the pulmonary function study performed by Dr. Smiddy is invalid for premature termination of air flow. However, Dr. Dahhan maintains that despite these invalid pulmonary function values, the obstruction of Mr. Parsons' breathing is severe enough to preclude his return to coal mine employment. Considering that Mr. Parsons left the mines over thirteen years ago and his obstructive defect is associated with normal lung volumes, Dr. Dahhan has ruled out pneumoconiosis as a cause of

Mr. Parsons' pulmonary impairment. Instead, Mr. Parsons' lengthy cigarette smoking habit is the etiology of the respiratory disability.

On August 8, 2000, Dr. Dahhan reviewed the pathological evidence associated with Mr. Parsons' death, which indicated the presence of simple coal workers' pneumoconiosis and the absence of complicated coal workers' pneumoconiosis. Dr. Dahhan continued to opine that Mr. Parsons suffered an obstructive ventilatory defect that precluded his return to coal mine employment due to his lengthy cigarette smoking habit. Mr. Parsons' death was due to cardiac arrhythmia and complication of coronary artery disease, which was unrelated to coal dust inhalation. In Dr. Dahhan's opinion, coal workers' pneumoconiosis did not cause, contribute to, or hasten Mr. Parsons' death.

On February 17, 2003, Dr. Dahhan again reviewed the radiographic and medical record which now included an autopsy report. Dr. Dahhan opined pathologic evidence established the presence of simple coal workers' pneumoconiosis. Mr. Parsons also had struggled with a mild obstructive ventilatory defect. In Dr. Dahhan's opinion, Mr. Parsons' death was caused by ventricular fibrillation, a complication of coronary artery disease, which was not caused by, related to, aggravated by, or contributed to by the inhalation of coal dust or coal workers' pneumoconiosis.

On March 8, 2004, Dr. Dahhan again reviewed Mr. Parsons' case, including several supplemental opinions. Dr. Dahhan stood firm with his prior conclusions. Pathology evidence showed Mr. Parsons had early simple coal workers' pneumoconiosis. Prior to his death, Mr. Parsons was totally disabled from a respiratory perspective due cigarette smoke induced chronic pulmonary obstruction. Likewise, his death and associated underlying cardiac problems were related to cigarette smoking and not coal dust exposure.

Dr. John A. Michos
(DX 29)

On March 31, 1997, Dr. Michos, board certified in pulmonary disease and internal medicine,[39] provided a medical opinion which considered Mr. Parsons' employment history, cigarette smoking history and several chest x-rays along with the complete medical reports from Dr. Taylor (September 23, 1996) and Dr. Dahhan (February 21, 1997). According to Dr. Michos, based on negative chest x-rays and arterial blood gas studies showing only mild hypoxemia, Mr. Parsons does not suffer from coal workers' pneumoconiosis. Mr. Parsons' dyspnea and moderate obstructive lung disease are more consistent with Mr. Parsons' chronic and continuing use of cigarettes. Dr. Michos believed Mr. Parsons is totally disabled and unable to return to coal mine employment. He attributed the total disability to significant heart disease.

Dr. Farid F. Zayed
(DX 53, DX 97, DX 100, DX 101, and DX 102)

[39]I take judicial notice of Dr. Michos' board certified and have attached the certification documentation.

On July 15, 1997, Dr. Zayed, board certified in cardiovascular disease,[40] performed a complete physical examination which revealed a deformed chest wall from a sternectomy. Mr. Parsons offered no complaints with respect to dyspnea, edema, wheezing or cough. In the final analysis, Dr. Zayed diagnosed COPD (chronic obstructive pulmonary disease) with coal workers' pneumoconiosis. The physician offered no commentary concerning the severity of Mr. Parsons' pulmonary impairment. On August 19, 1997, Dr. Zayed performed a second physical examination which revealed a stable cardiopulmonary system. The physician referred Mr. Parsons to Dr. Smiddy for evaluation of a lung nodule. On September 24, 1997, Dr. Zayed performed a final physical examination. The chest examination and heart examination were normal. Between October 1997 through March 1998, Dr. Zayed continued to periodically conduct follow-up evaluations for Mr. Parsons' chronic chest pain. On one visit, the physical examination indicated a clear chest.

On June 3, 1998, Dr. Zayed signed Mr. Parsons' death certificate indicating he passed away on June 2, 1998. Noting that a lung autopsy had been conducted, Dr. Zayed stated the immediate cause of death was ventricular fibrillation due to acute myocardial infarction associated with underlying coronary artery disease.

In an April 26, 1999 letter, Dr. Zayed noted that Mr. Parsons had been diagnosed with black lung and chronic lung disease. Mr. Parsons also had a cardiac condition and his death was possibly due to an acute myocardial infarction. However, in Dr. Zayed's opinion, "black lung with chronic lung disease, either could cause respiratory arrest and what could lead to a fatal ventricular arrhythmia or as a co-morbid condition that could contribute to this condition with a resultant worse outcome."

Dr. Joseph F. Smiddy
(DX 47, DX 53, and CX 1)

On July 26, 1991, Mr. Parsons experienced an acute myocardial infarction, a few days later upon presentation to the emergency room, he experienced coronary and respiratory failure. After being successfully revived, Mr. Parsons under underwent coronary artery bypass surgery on August 1, 1991. As part of the treatment, also on August 1, 1991, Dr. Smiddy, board certified in internal medicine, performed a pulmonary evaluation. According to his spouse, Mr. Parsons suffered from chronic lung disease and chronic respiratory impairment with coal workers' pneumoconiosis. The physician recorded a thirty-five year coal mine employment history and a long term cigarette smoking history. The chest examination revealed scattered squeaks, rales and rhonchi. Serial chest x-rays revealed minimal effusion atelectasis in the left lung base. An arterial blood gas on the ventilator produced non-qualifying values for total disability. In the final analysis, Dr. Smiddy diagnosed chronic obstructive pulmonary disease and pneumoconiosis along with cardiac disease and chronic back problems.

On September 9, 1997, Dr Smiddy performed a complete physical examination and chest x-ray. At the physical examination, Mr. Parsons complained of chest pains. Dr. Smiddy noted that an October 18, 1996 CT scan revealed a pleural mass in the left lower lung and that another physician suggested a mediastinoscopy and lung biopsy at that time. The physical examination

[40]I take judicial notice of Dr. Zayed's board certification and have attached the certification documentation.

revealed no rales, rubs or rhonchi. The chest x-ray revealed bilateral nodules with changes thought to be representative of pneumoconiosis plus old granulomatous disease, but the physician could not exclude the possibility of active process without comparison films. A second physical examination performed on September 19, 1997, revealed slightly decreased breath sounds with some harsh breath sounds. Dr. Smiddy diagnosed pneumoconiosis. At a third physical examination in December 1997, Mr. Parsons offered no pulmonary complaints. The physical examination revealed decreased breath sounds.

Dr. Gregory J. Fino
(DX 47, DX 51, DX 81, DX 137, DX 140, DX 149, DX 154, and DX 174)

On February 22, 1994, Dr. Fino, board certified in pulmonary disease and internal medicine, completed a comprehensive medical report after considering Mr. Parsons' employment history and all objective medical information in the official record. In regards to the chest x-ray evidence, Dr. Fino concluded that Mr. Parsons does not exhibit radiographic changes consistent with coal workers' pneumoconiosis. In particular, Dr. Fino disagreed with Dr. Sargent's interpretation of type "t" opacities as possibly consistent with pneumoconiosis. In the final analysis, Dr. Fino stated that Mr. Parsons does not suffer from coal workers' pneumoconiosis or any significant pulmonary impairment. According to Dr. Fino, from a respiratory perspective, Mr. Parsons could return to coal mine employment as a shuttle car operator, although coronary artery disease would prevent Mr. Parsons' return to coal mine employment.

In a deposition conducted on March 7, 1994, Dr. Fino considered additional medical information in the form of a complete physical examination performed by Dr. Dahhan before once again concluding that Mr. Parsons does not suffer from coal workers' pneumoconiosis and that from a respiratory perspective, Mr. Parsons could return to the coal mines to perform the moderate physical labor required of a shuttle car operator.

In a supplemental medical report completed on June 30, 1994, Dr. Fino considered a comprehensive medical report from Dr, Morgan and the complete physical examination performed by Dr. Dahhan. Once again, Dr. Fino concluded that Mr. Parsons does not suffer from coal workers' pneumoconiosis.

On February 23, 1998, Dr. Fino completed a comprehensive medical record review which considered fourteen interpretations of five chest x-ray films, two interpretations of a single CT scan, two pulmonary function studies, two arterial blood gas studies and three medical reports from the present claim along with an assortment of medical information from Mr. Parsons' October 1971 and February 1986 applications for federal black lung disability benefits. According to Dr. Fino, the pulmonary function studies performed on September 24, 1996 and February 21, 1997 are invalid for premature termination of exhalation and a lack of reproducibility. After considering this medical evidence, Dr. Fino stated Mr. Parsons does not suffer from a coal mine dust related pulmonary condition. He disagreed with Dr. Dahhan's diagnosis concerning the presence of pulmonary impairment. Because Dr. Fino thinks the February 1997 pulmonary function tests are invalid, he asserts Dr. Dahhan had no objective medical evidence upon which to base his conclusion. According to Dr. Fino, Mr. Parsons is not

disabled due to any respiratory impairment. In the event Mr. Parsons did have a pulmonary defect, while acknowledging that pneumoconiosis may cause an obstructive lung defect, Dr. Fino believes the cigarette smoking would nevertheless be cause. To support his conclusion, Dr. Fino pointed out that the lung volume results on the pulmonary function tests were not consistent with black lung disease. Instead, Mr. Parsons' long term cigarette smoking habit is the cause of any pulmonary defect.

On August 16, 2000, Dr. Fino reviewed additional medical evidence including several pathology reports. Dr. Fino noted the lung biopsies indicated the presence of simple coal workers' pneumoconiosis. However, the record still did not contain valid objective medical evidence of a respiratory impairment. Likewise, Mr. Parsons was not totally disabled due to pneumoconiosis. Mr. Parsons' death was due to coronary artery disease. Several medical studies have shown no relationship between coal dust inhalation and coronary artery disease. Coal workers' pneumoconiosis did not hasten Mr. Parsons' death.

In an October 6, 2000 deposition, Dr. Fino again discussed his assessment of Mr. Parsons' pulmonary disease and death. Having reviewed Mr. Parsons' lifetime medical record, Dr. Fino had concluded Mr. Parsons did not have coal workers' pneumoconiosis and no objective medical evidence of a respiratory impairment existed. Following his death, a lung biopsy showed the presence of coal and silicotic nodules, microscopically establishing the presence of coal workers' pneumoconiosis and silicosis. Microscopic evaluation is more sensitive and can identify early pneumoconiosis even if it hasn't reached the level of radiographic visibility. Chest x-ray remain a good tool for diagnosing medical pneumoconiosis because when the opacities start to appear radiographically, the pneumoconiosis has reached "a level by which it could be impairing or disabling depending on degree." Since Mr. Parsons suffered a sudden cardiac death caused by a myocardial infarction or cardiac arrhythmia, Dr. Fino disagrees with Dr. Zayed's opinion that Mr. Parsons' COPD and coal workers' pneumoconiosis could have caused his cardiac problems. While severe coal workers' pneumoconiosis might be considered a co-morbid condition, Mr. Parsons' pneumoconiosis was not at that level. Mr. Parsons' pneumoconiosis was "subclinical, meaning that you can diagnose it under the microscope, but two sensitive radiographic tests, a chest x-ray and a more sensitive CT scan, still did not pick it out." For coal workers' pneumoconiosis to have been a factor in Mr. Parsons' death, it must have been causing a lung function abnormality. The amount of Mr. Parsons' pneumoconiosis "was adding zero to this man's overall medical picture in any way."

On February 26, 2003, having reviewed the medical record, including hospitalization records from 1991 and 1995, Dr. Fino did not change any of his previous opinions.

On July 29, 2003, after reviewing Dr. Molony's assessment, Dr. Fino found no reason to change his opinions about Mr. Parsons' pulmonary condition and death. The new information did not provide any evidence that coal dust inhalation had contributed to or hastened Mr. Parsons' death.

Dr. James R. Castle
(DX 52, DX 58, DX 81, DX 137, DX 150, DX 153, DX 174, EX 4)

On February 25, 1998, Dr. Castle, board certified in pulmonary disease and internal medicine, completed a comprehensive medical record review after considering chest x-rays, CT scans, pulmonary function studies, arterial blood gas studies and medical reports from August 1968 through February 1997. According to Dr. Castle, Mr. Parsons does not suffer from coal workers' pneumoconiosis. In particular, he noted the absence of any finding of pneumoconiosis in the more accurate CT scans. Dr. Castle observed that Mr. Parsons may suffer from a mild to moderate obstructive airways disease, with some degree of reversibility. This mild to moderate obstructive impairment is caused by chronic cigarette smoking. Because Dr. Castle considered Mr. Parsons' recent pulmonary function tests in September 1996 and February 1997 to be technically invalid, he declined to offer any definite conclusions concerning the severity of Mr. Parsons' respiratory impairment, instead stating that it is "possible" that Mr. Parsons is disabled as a result of a moderate restrictive pulmonary impairment. However, the obstructive airways disease was totally unrelated to Mr. Parsons' coal mine employment or exposure to coal dust. In addition, Mr. Parsons was certainly totally disabled due to other health conditions including coronary artery heart disease, hypertension, diabetes, and cervical disc disease.

After considering additional medical evidence, including a pulmonary function study performed on September 19, 1997, Dr. Castle indicated in a March 1998 deposition that Mr. Parsons does not suffer from coal workers pneumoconiosis. According to Dr. Castle, Mr. Parsons is disabled due to his moderate obstructive airways diseases. However, based on all the medical information, Dr. Castle does not believe Mr. Parsons has pneumoconiosis. In particular, while recognizing that "legal" pneumoconiosis is a progressive disease, Dr. Castle indicated that in medical terms once a person leaves the coal mines and exposure to coal dust, pneumoconiosis does not advance significantly. Dr. Castle observed that CT scans are more accurate than x-rays in detecting pneumoconiosis. However, he added that pneumoconiosis may still exist even in the absence of radiographic evidence. That is, pathologic evidence may indicate the presence of coal workers' pneumoconiosis that did not cause any clinical symptoms of pneumoconiosis. Finally, based on his review of the record, Dr. Castle concluded Mr. Parsons' chronic obstructive pulmonary disease stemmed from tobacco smoke.

On August 23, 2000, Dr. Castle reviewed additional medical evidence, which included Mr. Parsons' death certificate and various lung biopsy reports. The pathology reports showed the presence of minimal coal workers' pneumoconiosis. However, Mr. Parsons' pneumoconiosis was "too minimal to have caused him any impairment during life and certainly did not play any role in his demise." Mr. Parson died due to cardiac arrhythmia associated with an acute myocardial infarction and atherosclerotic cardiovascular disease.

In an October 3, 2000 deposition, Dr. Castle indicated the presence of pathologic evidence of coal workers' pneumoconiosis does not alter his opinion that Mr. Parsons was not totally disabled due to coal workers' pneumoconiosis. The negative chest x-rays and CT scan findings "are actually reinforced by the fact that he had very minimal simple pathological evidence." Additionally, contrary to Dr. Zayed's assertion, minimal coal workers'

pneumoconiosis does not cause cardiac arrhythmia. For coal workers' pneumoconiosis to cause death, it must be "quite severe." Mr. Parsons' coal workers' pneumoconiosis was so minimal it didn't show up on chest x-rays. Mr. Parsons suffered a "clearly a cardiac death related to coronary artery disease and acute myocardial infarction."

On March 4, 2003, Dr. Castle again reviewed the medical record and additional hospitalization records. The physician remained convinced that Mr. Parsons had pathologic evidence of "minimal, simple coal workers' pneumoconiosis" that "did not cause him any respiratory impairment during his life." Mr. Parsons' pulmonary problems were entirely due to his extensive tobacco smoking habit which led to a moderate obstructive pulmonary impairment.

On July 28, 2003, after reviewing Dr. Molony's assessment, Dr. Castle opined the physician's opinion was not "substantiated or corroborated by any additional objective medical data or evidence." Pathologically, Mr. Parsons had "minimal, simple coal workers' pneumoconiosis." Most reviewers and pathologists agreed the pathologic coal workers' pneumoconiosis was of "insufficient severity to have caused him any impairment during life and did not contribute to his death." The coal workers' pneumoconiosis was of insufficient severity to produce x-ray and CT scan abnormalities. Mr. Parsons had severe coronary artery disease that required a coronary artery by-pass. Eventually, Mr. Parsons suffered a myocardial infarction and died due to ventricular fibrillation.

In a March 2004 deposition, Dr. Castle expressed his disagreement with the conclusions by Dr. Zayed and Dr. Molony that lung disease contributed to Mr. Parsons' death. Dr. Castle emphasized that Mr. Parsons had only minimal simple coal workers' pneumoconiosis that was not even detectable by chest x-rays. At the most, Mr. Parsons only suffered a mild degree of obstruction associated with his cigarette smoking and some degree of restriction due to his sternectomy. Mr. Parsons was not totally disabled due to coal workers' pneumoconiosis. According to Dr. Castle, "Mr. Parsons had very serious coronary artery disease, and his death was totally related to that process." Mr. Parsons' heavy cigarette smoking "very significantly" contributed to his death.

Dr. W.K.C. Morgan
(DX 47, DX 54, DX 140, DX 148, DX 81, and EX 1

On February 18, 1994, Dr. Morgan conducted a medical record review by considering all chest x-ray interpretations, pulmonary function studies, arterial blood gas studies and medical reports in the official record. Based on the objective medical evidence, Dr. Morgan stated that Mr. Parsons does not suffer from coal workers' pneumoconiosis. In particular, Dr. Morgan disagreed with Dr. Sargent's medical opinion that Mr. Parsons' "t" opacities could be caused by coal dust exposure. According to Dr. Morgan, coal dust exposure never causes type "t" opacities. The physician explained Mr. Parsons' pulmonary impairment as a combination of chronic cigarette smoking and coronary artery bypass surgery. In the final analysis, Dr. Morgan stated that Mr. Parsons could not return to coal mine employment because of cervical spinal disease, coronary artery disease and diabetes.

On July 4, 1994, Dr. Morgan provided a supplemental medical report after considering a consultative medical report from Dr. Fino and a physical examination, chest x-ray, pulmonary

function study and arterial blood gas study performed by Dr. Dahhan. Once again, Dr. Morgan stated that Mr. Parsons does not suffer from coal workers' pneumoconiosis. Although the retired coal miner is totally disabled and cannot return to coal mine employment, Dr. Morgan indicated Mr. Parsons' total disability is not related to coal dust exposure.

In a comprehensive medical record review completed on March 2, 1998, Dr. Morgan considered all the medical evidence from Mr. Parsons' June 1996 application for federal black lung disability benefits along with several chest x-rays, medical reports and depositions from the earlier February 1986 claim. According to Dr. Morgan, Mr. Parsons suffers from a "mild to moderate restrictive impairment and probably, but not definitely, [a] mild obstructive impairment." The mild to moderate restrictive impairment is caused by Mr. Parsons' coronary artery disease and subsequent sternectomy. Any obstructive pulmonary impairment is caused by Mr. Parsons' chronic cigarette smoking. According to Dr. Morgan, Mr. Parsons is totally disabled from coronary artery disease and back problems. However, if Mr. Parsons did not suffer from coronary artery disease with the complications of a sternectomy, Dr. Morgan believed Mr. Parsons would retain sufficient respiratory capacity to return to the coal mine.

On August 12, 2000, Dr. Morgan reviewed Mr. Parsons' death certificate and recent pathology reports and concluded Mr. Parsons had mild coal workers' pneumoconiosis and silicosis. Significantly, neither pneumoconiosis nor silicosis were evident on the chest x-rays, "indicating that these were relatively mild and would not in any way interfere with his capacity to work in the mines." The pathology evidence of emphysema also supported his prior conclusion that Mr. Parsons had a mild to moderate obstruction. Dr. Morgan diagnosed "very mild" coal workers' pneumoconiosis and "mild" silicosis. Neither condition affected Mr. Parsons' lung functions. Mr. Parsons retained the respiratory reserve to return to coal mine employment. Any impairment he suffered was due to prolonged cigarette smoking. Coal dust did not play any role in hastening Mr. Parsons' death.

On February 17, 2003, Dr. Morgan again discussed Mr. Parsons' pulmonary condition in light of his medical record and post-mortem evidence. Noting that Mr. Parsons' developed respiratory problems long after he left coal mining, the physician indicated a possible cause was his developing overweight condition. Mr. Parsons' limited obstructive impairment was associated with his "prolonged" cigarette smoking habit and would not have stopped him from returning to coal mine employment. At the same time, Mr. Parsons was totally disabled to cardiac disease and other health problems. The post-mortem evidence also indicated that coal dust inhalation did not cause his breathing problems. The autopsy and microscopic evidence showed the presence of mild CWP (coal workers' pneumoconiosis), consisting of a few macules, and silicotic nodules, attributable to his work as a shuttle car operator and the associated use of sand for traction on the rails. According to Dr. Morgan, "neither CWP nor silicosis was evident in the chest x-rays indicating that neither coal dust nor silicosis were present in sufficient quantity to interfere with Mr. Parson's capacity to work in the mines."

On March 5, 2004, Dr. Morgan reviewed several supplemental medical opinions and saw no need to change his opinions. Although Mr. Parsons had a pulmonary impairment due to cigarette smoking, the minimum coal dust and silica deposits in Mr. Parsons' lungs in no way

affected his ability to work in coal mines. Coal dust played no role in Mr. Parsons' disability or death.

Dr. Kanti D. Patel
(DX 98)

On June 9, 1998, Dr. Patel, a board certified pathologist,[41] examined the lung tissue slides from Mr. Parsons' autopsy for black lung disease. The physician noted the pleural surface was gray-brown to black due to black pigment deposit. The physician diagnosed diffuse pulmonary emphysema, interstitial fibrosis and black pigment deposition.

Dr. Jeffrey Kahn
(DX 101 and DX 102)

On July 19, 1999, Dr. Kahn, a board certified pathologist,[42] reviewed Mr. Parsons' lung tissue slides. He noted the presence of moderate pulmonary emphysema and "small quantities of coal dust" and several small coal macules indicative of coal workers' pneumoconiosis. No coal dust nodules were present. Dr. Kahn also found multiple silicotic nodules and one confluence of silicotic nodules, representing moderate silicosis.

Dr. Richard L. Naeye
(DX 99, DX 135, and DX 174)

On August 29, 1999, Dr. Naeye, a board certified pathologist, microscopically examined the lung tissue specimens from Mr. Parsons' autopsy. He also reviewed the death certificate, the autopsy report, and a few chest x-ray interpretations. In the lung tissue, Dr. Naeye found a small amount of black pigment deposits measuring less than one millimeter which were anthracotic macules. He also noted old silicotic nodules less that 8 mm in diameter. A few of the silicotic lesions were grouped in a mass; however the macronodules did not represent complicated pneumoconiosis. Dr. Naeye also noted the presence of centrilobular emphysema. The physician diagnosed, mild simple coal workers' pneumoconiosis. He noted that since the chest x-rays did not show the presence of pneumoconiosis, it was overall mild in severity. Specifically, the coal workers' pneumoconiosis was "too limited to have caused any disability or contributed in any way" to Mr. Parsons' death. Mr. Parsons died due to complications of arteriosclerotic coronary artery disease.

On November 18, 1999, Dr. Naeye again evaluated the pathology lung tissue specimens. In the ten lung specimens, Dr. Naeye observed: a) mild to moderate black pigment deposits, measuring 0.1 to 0.8 mm in diameter; and, b) several areas of old anthrasilicotic macronodules which represented a collection of old lesions. The latter finding was not complicated pneumoconiosis. Dr. Naeye also noted the presence of centrilobular emphysema and focal emphysema, which accounted for less than 1% of the overall emphysema in the lung tissue.

[41]I take judicial notice of Dr. Patel's board certification and have attached the certification documentation.

[42]I take judicial notice of Dr. Kahn's board certification and have attached the certification documentation.

Based on his observations, Dr. Naeye diagnosed mild simple coal workers' pneumoconiosis. Since the preponderance of the chest x-ray interpretations were negative for pneumoconiosis, Dr. Naeye believed its severity was "mild." Whatever Mr. Parsons' pulmonary impairment may have been, it was due to his cigarette smoking history. According to Dr. Naeye, "His CWP was too mild to have caused disability or have hastened or contributed to his death."

In a January 11, 2000 deposition, Dr. Naeye discussed his pathology determinations and review of Mr. Parsons' medical record. Mr. Parsons had 33 years of coal mine employment and smoked up to a pack and a half of cigarettes a day for 36 years. In the lung tissue slides, two types of coal workers' pneumoconiosis were present. First, Dr. Naeye detected a small to moderate amount black pigment deposits, or anthracotic micronodules, of less than one millimeter in diameter. These micronodules would not have cause any abnormalities in lung function. Second, Dr. Naeye found six areas of black deposits, measuring 6 to 13 millimeters. These macronodules had an old silicotic origin and did not represent complicated pneumoconiosis. Dr. Naeye explained, "Those old silicotic macronodules can arise in adjacent areas and eventually come in contact with each other . . . and cause a conglomerate mass, but those lesions in no way resembles complicated coal workers' pneumoconiosis." Complicated coal workers' pneumoconiosis involves an immunologic process with action associated with inflammatory cells. The process destroys blood vessels and the center of the coal workers' pneumoconiosis lesions become necrotic and fill with black liquid. Consequently, when a chest x-ray shows a large pulmonary mass, it is "absolutely imperative" to ascertain whether the opacity represents a conglomerate mass of old fibrotic micronodules that "finally grew together" or the active process of complicated pneumoconiosis. Dr. Naeye also noted that the lung tissue samples usually are not representative of the entire lungs since the autopsy prosecutor will usually select the blackest areas for dissection and evaluation. While that approach makes sense analytically, the selective nature of the procedure is "misleading in terms of its severity." In Dr. Naeye's opinion, Mr. Parsons' death was caused by an acute myocardial infarction, unrelated to his coal dust exposure. Instead, Mr. Parsons' coronary artery disease and long term cigarette smoking habit were contributing factors. The coal workers' pneumoconiosis lesions in Mr. Parsons' lungs were "too small and too few" to have caused any lung impairment or hastened his death. Since Mr. Parsons suffered cardiac arrhythmia, Dr. Naeye specifically disagreed with Dr. Zayed's opinion that Mr. Parsons may have suffered respiratory arrest associated with coal workers' pneumoconiosis.

In July 17, 2003, in response to Dr. Molony's opinion that lung disease contributed to Mr. Parsons' death, Dr. Naeye noted the pathologic evidence of anthracotic macules and several old anthracosilicotic macules only met the minimum standard for a diagnosis of simple coal workers' pneumoconiosis. Additionally, the chest x-rays and other pulmonary tests did not "produce evidence of severe enough CWP to have caused disability or contribute to this man's death."

Dr. P. Raphael Caffrey
(DX 81, DX 135, and EX 2)

On December 14, 1999, Dr. Caffrey, board certified in anatomic and clinical pathology, reviewed Mr. Parsons radiographic and medical records, which included the pathology reports of

Dr. Kahn and Dr. Patel. Dr. Caffrey also microscopically examined the lung tissue slides. Dr. Caffrey noted the presence of: a) a mild to moderate amount of anthracotic pigment "with a rare macule," b) a few silicotic nodules, ranging singly from 1 millimeter to confluent nodules, measuring 8 mm; and, c) a mild degree of pulmonary emphysema. Dr. Caffrey found no evidence of complicated pneumoconiosis. Based on his evaluation, Dr. Caffrey diagnosed moderate simple silicosis, mild simple coal workers' pneumoconiosis, and mild pulmonary emphysema. The degree of simple silicosis and coal workers' pneumoconiosis would not have hastened Mr. Parsons' death. Mr. Parsons' main pulmonary problems were due to his two pack a day cigarette smoking habit that spanned 30 to 40 years. His main health issue was coronary artery disease.

On February 19, 2003, after again considering the medical record and post-mortem evidence, Dr. Caffrey opined that Mr. Parsons suffered a totally disabling pulmonary and respiratory impairment. That impairment was caused by Mr. Parsons smoking two packs of cigarettes a day for 40 years. Coal workers' pneumoconiosis did not contribute to the impairment because Mr. Parsons only had "a very mild degree of simple CWP." According to Dr. Caffrey, Mr. Parsons had "pathologic CWP, not clinical CWP."

On March 6, 2004, after reviewing supplemental medical opinions and reports, Dr. Caffrey made no changes in his assessments about Mr. Parsons' pulmonary condition and death. In response to Dr. Molony's statement that Dr. Taylor's treatment notes established COPD and pneumoconiosis, Dr. Caffrey observed that to the contrary almost all the dual qualified radiologists' interpretations were actually negative for pneumoconiosis. Additionally, Dr. Molony did not specifically identify "one iota of objective data to support" his opinion that lung disease was a contributing factor in Mr. Parsons' death. Significantly, Mr. Parsons' pathologic silicosis and coal workers' pneumoconiosis did not display clinical signs or symptoms.

Dr. Patrick Molony
(DX 171, DX 172, CX 1)

On May 26, 2003, Dr. Molony indicated that he had reviewed Dr. Taylor's treatment notes for Mr. Parsons. The record "clearly documents a diagnosis of COPD and pneumoconiosis." He also noted that Dr. Kahn's lung biopsy confirmed the presence of pulmonary emphysema and pneumoconiosis. Based on this information, Dr. Molony concluded "Mr. Parsons had serious cardiac disease which caused death but lung disease was a contributing factor."

On August 14, 2005, Dr. Molony indicated the medical record had not changed and he expressed his belief, "this has been dragging on for a very long time."

Dr. David M. Rosenberg
(EX 5)

On January 3, 2005, Dr. Rosenberg, board certified in pulmonary disease, internal medicine, and occupational medicine, conducted an extensive medical record review, covering the radiographic interpretations, multiple pulmonary evaluations, dozens of medical opinions,

and autopsy/pathology reports. Dr. Rosenberg noted that Mr. Parsons had mined coal for over 30 years and had a long-term and heavy cigarette smoking history. His medical history included coronary artery disease, a myocardial infarction and subsequent coronary artery by-pass, and surgical removal of his sternum due to a post-operative infection. During the course of Mr. Parsons' life, the preponderance of the clinical and radiographic record, including CT scans, was insufficient to establish the presence of pneumoconiosis. However, upon his passing, subsequent pathology examination identified the presence of mild simple coal workers' pneumoconiosis. Up through 1990, Mr. Parsons did not have a totally disabling pulmonary impairment. However, after 1990, "Mr. Parsons from a pulmonary perspective was considered disabled." According to Dr. Rosenberg, "Mr. Parsons' extensive and continued smoking with abnormal chest wall mechanics are responsible for any [pulmonary] obstruction after 1990." Dr. Rosenberg opined that Mr. Parsons' total respiratory disability was not due to coal dust because the pathologically identified pneumoconiosis did not cause or contribute to the impairment. The physician stressed that although simple pneumoconiosis was identified pathologically after Mr. Parsons' death, coal workers' pneumoconiosis was "not apparent clinically [or] even diagnosable on a CAT scan of the chest." Mr. Parsons' death was caused by ventricular fibrillation related to an acute myocardial infarction with underlying atherosclerotic heart disease. Mr. Parsons' pneumoconiosis did not contribute or exacerbate Mr. Parsons' heart problem. To have a discernable effect, a lung disorder must cause hypoxemia, leading to diminished oxygen delivery to the heart. In Mr. Parsons' case, while arterial blood gas studies showed a mild reduction in blood oxygenation, his blood oxygen was "well above 90%" or a $_{p}O_{2}$ greater than 60 As a result, Dr. Rosenberg concluded, "there is no reason to suspect in any fashion that his underlying lung disease caused or contributed to a decreased oxygen content within the blood, contributing to fatal arrhythmia." Instead, Mr. Parsons' fatal myocardial infarction was strictly related to his heart disease, which was unrelated to his exposure to coal dust. His death was not caused or hastened in any manner by coal dust exposure or the presence coal workers' pneumoconiosis.

Dr. Kirk E. Hippensteel
(EX 6)

On February 3, 2005, Dr. Hippensteel, board certified in pulmonary disease and internal medicine, conducted an extensive medical record review, covering the radiographic interpretations, multiple pulmonary evaluations, dozens of medical opinions, and autopsy/pathology reports. Finding the pathology reports more probative than the radiographic record, Dr. Hippensteel concluded Mr. Parsons had very mild simple coal workers' pneumoconiosis "pathologically." Concerning respiratory function, Mr. Parsons developed a "significant lung impairment" long after he left coal mining and while he continued to smoke cigarettes. Additionally, his pulmonary impairment was partially reversible. Further, the pathology findings of minimal simple coal workers' pneumoconiosis and no complicated pneumoconiosis "are usually associated with very little pulmonary impairment." These considerations "favor cigarette smoking as a cause of impairment." Mr. Parsons' "coronary artery disease and secondary pulmonary function effects from complications of surgery for the coronary artery disease lead to increased chest symptoms that were not related specifically to intrinsic lung disease." On the other hand, coronary artery disease is related to cigarette smoking. Mr. Parsons died a cardiac death unrelated to lung disease. Mr. Parsons' mild, simple coal workers' pneumoconiosis did not cause, contribute to, or hasten his death.

Discussion

On the issue of whether Mr. Parsons was totally disabled due to a pulmonary impairment, the following doctors did not render an opinion: Dr. Miller, Dr. Michos,[43] Dr. Zayed, Dr. Smiddy, Dr. Patel, Dr. Kahn, Dr. Naeye, Dr. Molony, and Dr. Hippensteel.[44] The remaining physicians who considered Mr. Parsons' pulmonary condition were nearly evenly split on total disability. Dr. Fleenor, Dr. Paranthaman, Dr. Abrahams, Dr. Fino, and Dr. Morgan did not believe Mr. Parsons suffered a totally disabling respiratory impairment. To the contrary, Dr. Nash, Dr Sargent, Dr. Taylor, Dr. Dahhan, Dr. Castle, Dr. Caffrey, and Dr. Rosenberg concluded Mr. Parsons was totally disabled due to a pulmonary impairment. Due to this conflict in medical opinion, I must first assess the relative probative value of each respective opinion in terms of documentation, reasoning, medical qualifications, and treating physician status.

Regarding the first probative value consideration, documentation, a physician's medical opinion is likely to be more comprehensive and probative if it is based on extensive objective medical documentation such as radiographic tests and physical examinations. *Hoffman v. B & G Construction Co.*, 8 B.L.R. 1-65 (1985). In other words, a doctor who considers an array of medical documentation that is both long (involving comprehensive testing) and deep (includes both the most recent medical information and past medical tests) is in a better position to present a more probative assessment than the physician who bases a diagnosis on a test or two and one encounter.

The second factor affecting relative probative value, reasoning, involves an evaluation of the connections a physician makes based on the documentation before him or her. A doctor's reasoning that is both supported by objective medical tests and consistent with all the documentation in the record, is entitled to greater probative weight. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). Additionally, to be considered well reasoned, the physician's conclusion must be stated without equivocation or vagueness. *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91 (1988).

Third, a physician who is board-certified in the field of pulmonary disease and who has extensive experience in this area may be accorded greater deference because of his or her expertise. *Clark v. Karst-Robbins Coal Co.*, 12 BLR 1-149 (1989) (en banc); *Fields v. Island Creek Coal Co.*, 10 BLR 1-19 (1987); *Burns v. Director, OWCP*, 7 BLR 1-597 (1984).

Fourth, according to 20 C.F.R. § 718.104 (d), in evaluating medical opinion, an administrative law judge must consider the relationship between the claimant and any treating physician. Depending on the duration, frequency, and extent of the treatment, the opinion of a physician who provided treatment for pulmonary concerns may be entitled to more probative

[43]Dr. Michos determined Mr. Parsons had a moderate obstructive pulmonary impairment. However, he addressed Mr. Parsons' total disability in terms of Mr. Parsons' heart disease and did not specifically address whether the moderate pulmonary impairment, standing alone, was totally disabling.

[44]Dr. Hippensteel opined that Mr. Parsons had a significant pulmonary impairment but did not specifically indicate whether the impairment was totally disabling.

weight than the assessment of a non-treating physician. At the same time, no presumption of greater probative weight exists merely based on a physician providing treatment. *See Consolidation Coal Co. v. Director, OWCP [Held],* 314 F.3d 184 (4th Cir. 2002).

With these principles in mind, I first note that the earlier medical opinions understandably are not as well documented as the more recent assessments. In particular, based on Dr. Sargent's and Dr. Rosenberg's observations about the pulmonary consequences of the 1991 sternectomy, the physicians who evaluated Mr. Parsons' pulmonary capacity before 1991 where not aware of the subsequent chest wall surgery and readily identifiable drop in his FEV_1 values in his pulmonary function tests. As a result, since their assessments were based on incomplete and dated documentary basis, the opinions of Dr. Fleenor (1979), Dr. Paranthaman (1987-1988), Dr. Abrahams (1990), and Dr. Nash (1990) have diminished probative value on whether Mr. Parsons had the respiratory capacity to return to coal mining.

The remaining opinions are documented and reasoned concerning the issue of total disability. Based on their review of the objective medical tests and pathology reports, Dr. Fino and Dr. Morgan, board certified pulmonologists, concluded Mr. Parsons retained the pulmonary capacity to return to coal mining. On the other hand, reviewing the same objective medical tests and autopsy results, Dr. Dahhan, Dr. Castle, Dr. Caffrey, and Dr. Rosenberg, similarly well qualified pulmonary physicians, concluded Mr. Parsons was totally disabled due to a pulmonary impairment prior to his death. Similarly, though they didn't have the pathology results before them, Dr. Sargent, also a board certified pulmonary expert, and Dr. Taylor, Mr. Parsons' long term treating physician, determined the objective test results, pulmonary examinations, and treatments established that Mr. Parsons' pulmonary condition precluded his return to coal mining. Since these respective assessments have essentially the same probative value concerning the extent of disability, I find pulmonary non-disability conclusions of Dr. Fino and Dr. Morgan are outweighed the consensus of Dr. Dahhan, Dr. Castle, Dr. Caffrey, Dr. Rosenberg, Dr. Sargent, and Dr. Taylor that Mr. Parsons had a totally disabling pulmonary impairment. Accordingly, through the preponderance of probative medical opinion total disability is established under 20 C.F.R. § 718.204 (b) (2) (iv).

Total Disability Due to Coal Workers' Pneumoconiosis

Since the first three element of entitlement have been established, the payment of black lung disability benefits will be warranted if the preponderance of probative medical evidence demonstrates that the coal workers' pneumoconiosis in Mr. Parsons' lungs played a role in his pulmonary insufficiency to return to coal mine employment. Significantly, proof that a claimant has a totally disabling pulmonary disease does not by itself establish the impairment is due to pneumoconiosis. Pursuant to 20 C.F.R. § 718.204 (c) (1), absent a favorable regulatory presumption,[45] a claimant must demonstrate that pneumoconiosis was a substantially contributing cause of his total disability by showing the disease: 1) had a material, adverse effect

[45]20 C.F.R. § 718.305 (if complicated pneumoconiosis is present, then there is an irrebuttable presumption the claimant is totally disabled due to pneumoconiosis); 20 C.F.R. § 718.305 (for claims filed before January 1, 1982, if the miner has fifteen years or more of coal mine employment, there is a rebuttable presumption that total disability is due to pneumoconiosis); and, 20 C.F.R. § 718.306 (a presumption exists when a survivor files a claim prior to June 30, 1982).

on his respiratory or pulmonary condition; or, 2) materially worsened a totally disabling respiratory impairment caused by a disease or exposure unrelated to pneumoconiosis. Additionally, 20 C.F.R. § 718.204 (c) (2) mandates that "the cause or causes of a miner's total disability shall be established by means of a physician's documented and reasoned medical report."

On the contribution coal workers' pneumoconiosis made to Mr. Parsons' pulmonary impairment, the physicians again either rendered no opinion or presented conflicting opinions. Specifically, Dr. Fleenor, Dr. Miller, Dr. Zayed, Dr. Smiddy, Dr. Patel, Dr. Kahn, and Dr. Molony did not address total disability etiology. Dr. Nash and Dr. Taylor believed coal workers' pneumoconiosis contributed to Mr. Parsons' pulmonary impairment. On the other hand, Dr. Paranthaman, Dr. Abrahams, Dr. Sargent, Dr. Michos, Dr. Dahhan, Dr. Fino, Dr. Castle, Dr. Morgan, Dr. Naeye, Dr. Caffrey, Dr. Rosenberg, and Dr. Hippensteel identified cigarette smoking and on occasion, chest wall deformity, and not coal dust exposure as the cause of Mr. Parsons' pulmonary problems. Based on this conflict of medical opinion, I once again return to probative value assessment.

Dr. Paranthaman's assessment on causation has diminished probative due to reasoning and documentation concerns. While Dr. Paranthaman attributed Mr. Parsons' chronic bronchitis to his cigarette smoking, he also conceded coal dust exposure could have aggravated the situation. However, that concession is somewhat equivocal and the physician failed to provide an explanation for his concession. More significantly, since he rendered the opinion in 1990, Dr. Paranthaman was unaware of the later pathology findings, which provided additional evidence about the nature and extent of Mr. Parsons' coal workers' pneumoconiosis.

Similarly, Dr. Abrahams' conclusion that Mr. Parsons' chronic bronchitis was due to his cigarette smoking has diminished probative weight for a documentation issue. Due to the dated nature of his evaluation, he was unaware of, and correspondingly unable to address, the presence of coal workers' pneumoconiosis in Mr. Parsons' lung tissue established by the pathology studies.

Dr. Nash's identification of coal workers' pneumoconiosis as the "majority" cause of Mr. Parsons' pulmonary impairment, rather than cigarette smoking, has diminished probative value since he based his conclusion on one pulmonary examination conducted in April 1990. Due to the date of his examination and sole consideration of its results, Dr. Nash's opinion has a documentary shortfall. Though he believed Mr. Parsons had coal workers' pneumoconiosis, Dr. Nash was unaware of the amount and nature of coal workers pneumoconiosis identified by several pathologists. Consequently, Dr. Nash's assessment is clearly not as well documented as other assessments based on all the medical evidence, including the pathology findings.

For the same documentary shortfall, Dr. Sargent's etiology conclusion has diminished probative value. Dr. Sargent believed Mr. Parsons' cigarette smoking and 1991 chest wall surgery caused his pulmonary impairment rather than the coal workers' pneumoconiosis which might be identifiable on the chest x-rays. Dr. Sargent based his assessment solely on medical evidence through 1994. Thus, due to the dated nature of his evaluation, Dr. Sargent did not consider the pathology findings.

Likewise, Dr. Michos' conclusion that Mr. Parsons' moderate obstructive lung disease was more consistent with his cigarette smoking has diminished probative value. In providing his assessment, Dr. Michos only considered the medical record through 1997 and was unaware of the 1998 autopsy results.

As Mr. Parsons' treating physician from 1988 through 1995, and having examined Mr. Parsons in January 1997, Dr. Taylor was well positioned to present a probative causation opinion. However, despite his treating physician status, Dr. Taylor's conclusion that coal workers' pneumoconiosis contributed to Mr. Parsons' total disability has diminished probative value for incomplete documentation. Similar to Dr. Paranthaman, Dr. Abrahams, Dr. Nash, Dr. Sargent, and Dr. Michos, Mr. Parsons' treating physician, Dr. Taylor, was unaware of the 1998 pathology findings of mild simple coal workers' pneumoconiosis.

Based on two pulmonary examinations and a medical record review, which included the pathology findings, Dr. Dahhan identified cigarette smoke as the sole cause of Mr. Parsons' chronic obstructive impairment. Although Dr. Dahhan's consideration of the pathology reports renders his assessment better documented, he presented his assessment in a conclusive manner without explanation. Dr. Dahhan did not indicate how he was able to determine that the simple coal workers' pneumoconiosis found in Mr. Parsons' lung tissue did not play a role in his impairment. Consequently, due to insufficient reasoning, Dr. Dahhan's opinion has diminished probative value.

Upon completion of an extensive medical record review, which included the numerous pulmonary evaluations and the pathology results, Dr. Fino presented a probative, well documented and reasoned medical opinion. According to Dr. Fino, the "subclinical" pneumoconiosis found in Mr. Parsons' lung tissue did not rise to level to produce either radiographic or clinical symptoms. As a result, the pathologic pneumoconiosis did not have any adverse effect on Mr. Parsons' pulmonary functions. Instead, noting in particular normal lung volumes in the pulmonary function tests, which are inconsistent with damage caused by pneumoconiosis, Dr. Fino opined cigarette smoke was the cause of any pulmonary difficulties suffered by Mr. Parsons.

Following a comprehensive medical record review and consideration of the pathology findings, Dr. Castle presented a probative, well documented and reasoned medical opinion. Relying on the pathologic findings of only mild, simple coal workers' pneumoconiosis, noting the reversibility in the pulmonary function tests, and emphasizing the absence of radiographic evidence of coal workers' pneumoconiosis, Dr. Castle concluded Mr. Parsons' pneumoconiosis was of "insufficient severity" to have caused any respiratory impairment. Mr. Parsons' had an obstructive impairment due to his chronic cigarette smoking.

In a similar manner, Dr. Morgan conducted a medical record review and evaluated the pathology reports to form a probative, well documented and reasoned medical opinion. In Dr. Morgan's opinion, the "relatively mild" pneumoconiosis observed during the biopsy of Mr. Parsons' lung tissue, which was not evident in the chest x-rays was not present in sufficient quantity to have interfered with his breathing. On the other hand, the pathologic presence of

emphysema confirmed his earlier conclusion based on clinical symptoms that Mr. Parsons' obstructive impairment was caused by his chronic cigarette smoking.

In addition to conducting a medical record review, Dr. Naeye also microscopically examined Mr. Parsons' lung tissue. In a probative, well documented and reasoned medical opinion, Dr. Naeye explained the pneumoconiosis macules were "too small" and "too few" to have caused any lung impairment. Dr. Naeye believed his conclusion was corroborated by the preponderance of the chest x-ray evidence which was negative for pneumoconiosis, indicating the pathologic pneumoconiosis was "too limited" to have caused any disability. Noting the presence of emphysema and other clinical data, Dr. Naeye concluded Mr. Parsons' obstructive impairment was caused by his chronic cigarette smoking.

Based on his review of medical record and evaluation of the lung tissue samples, Dr. Caffrey reached a probative, well documented and reasoned conclusion that Mr. Parsons' "very mild degree" of coal workers' pneumoconiosis did not contribute to his pulmonary impairment. Noting the absence of clinical symptoms of pneumoconiosis, and minimal pathologic pneumoconiosis, Dr. Caffrey attributed Mr. Parsons' pulmonary impairment to his extensive cigarette smoking history.

After a comprehensive review of the medical record and pathology evaluations, Dr. Rosenberg presented a probative, well documented and reasoned medical opinion. Although pathology studies found very mild pneumoconiosis, the preponderance of the clinical and radiographic evidence was insufficient to diagnose coal workers' pneumoconiosis. Consequently, Dr. Rosenberg opined the pathologic pneumoconiosis did not contribute to Mr. Parsons' pulmonary disability. Mr. Parsons' pulmonary obstructive defect was due to extensive and continued cigarette smoking and abnormal chest wall mechanics.

Finally, Dr. Hippensteel reached a probative, well documented and reasoned medical opinion upon completion of his review of the entire medical evidence in this case, including the pathology findings. The pathologic presence of "very mild simple coal workers' pneumoconiosis," which is usually associated with "very little pulmonary impairment," coupled with pulmonary test showing a partially reversible pulmonary impairment, "favored" Mr. Parsons' extensive cigarette smoking as the cause of his pulmonary impairment.

In summary, due to limited documentation associated with the absence of any consideration of the pathology findings, the conclusions by Dr. Paranthaman, Dr. Abrahams, Dr. Nash, Dr. Sargent, Dr. Michos, and Dr. Taylor have diminished probative value. Since Dr. Dahhan presented a causation conclusion without explanation, his opinion on the cause of total disability is not well reasoned and has diminished probative value. In contrast, the well documented and reasoned opinions of Dr. Fino, Dr. Castle, Dr. Morgan, Dr. Naeye, Dr. Caffrey, Dr. Rosenberg, and Dr. Hippensteel are more probative on the adverse pulmonary effects of the coal workers' pneumoconiosis in Mr. Parsons' lungs. Their more probative consensus convincingly establishes that coal workers' pneumoconiosis did not cause or contribute to Mr. Parsons' pulmonary impairment. As result, the preponderance of the more probative medical opinion does not establish that Mr. Parsons' totally disabling pulmonary impairment was due to coal workers' pneumoconiosis under 20 C.F.R. § 718.204 (c). Correspondingly, the record

before me does not establish the final requisite element of entitlement for black lung disability benefits.

Summary

Through stipulations, pathological evidence, and presumptions, the first three elements of entitlement for black lung disability benefits have been established: Mr. Parsons had coal workers' pneumoconiosis and suffered a totally disabling pulmonary impairment. However, based on the preponderance of the more probative opinions by physicians who evaluated the entire medical record, including the pathological findings of mild, simple coal workers' pneumoconiosis, I find coal workers' pneumoconiosis did not cause or contribute to Mr. Parsons' total pulmonary impairment.

Mrs. Parsons' Survivor Claim –
Issue #1 – Modification

Any party to a proceeding may request modification at any time before one year from the date of the last payment of benefits or at any time before one year after the denial of a claim. 20 C.F.R. § 725.310 (a). Upon the showing of a "change in conditions" or a "mistake in a determination of fact" the terms of an award or the decision to deny benefits may be reconsidered. 20 C.F.R. § 725.310. An order issued at the conclusion of a modification proceeding may terminate, continue, reinstate, increase or decrease benefit payments or award benefits.

Since the present modification relates to Mrs. Parsons' survivor claim, evaluation of the record for a change in conditions is not warranted.[46] Instead, the focus in modification proceedings in a survivor claim concerns a mistake of fact analysis. In *O'Keefe v. Aerojet-General Shipyards, Inc.*, 404 U.S. 254, 257 (1971), the United States Supreme Court indicated that an administrative law judge should review all evidence of record to determine if the original decision contained a mistake in a determination of fact. In considering a motion for modification, the administrative law judge is vested "with broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." *See also Jessee v. Director, OWCP*, 5 F.3d 723 (4th Cir. 1993); *Director, OWCP v. Drummond Coal Co. (Cornelius)*, 831 F.2d 240 (11th Cir. 1987).

My determination of whether a mistake of fact occurred during the prior adjudication of Mrs. Parsons' survivor claim involves the four entitlement elements that a claimant must prove by a preponderance of the evidence to receive survivor benefits under the Act and 20 C.F.R. § 718.205 (a). The claimant bears the burden of establishing these elements by a preponderance of the evidence. If the claimant fails to prove any one of the requisite elements, the survivor claim for benefits must be denied. *Gee v. W. G. Moore and Sons*, 9 B.L.R. 1-4 (1986) and *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985).

[46]Since Mr. Parsons has passed away, there can be no change in conditions concerning his pulmonary condition since the denial of Mrs. Parsons' survivor claim.

First, the claimant must establish eligibility as a survivor. A surviving spouse may be considered eligible for benefits under the Act if she was married to, and living with, the coal miner at the time of his death, and has not remarried.[47]

Second, the claimant must prove the coal miner had pneumoconiosis.[48] "Pneumoconiosis" is defined as a chronic dust disease arising out of coal mine employment. The regulatory definitions include both clinical pneumoconiosis (the diseases recognized by the medical community as pneumoconiosis) and legal pneumoconiosis (defined by regulation as any chronic lung disease arising out of coal mine employment).[49] The regulation further indicates that a lung disease arising out of coal mine employment includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."[50] As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

Third, once a determination has been made that a miner had pneumoconiosis, it must be determined whether the coal miner's pneumoconiosis arose, at least in part, out of coal mine employment.[51]

Fourth, the surviving spouse has to demonstrate the coal miner's death was due to pneumoconiosis.[52]

In Mrs. Parsons' case, the first three elements of entitlement have been established through stipulations, regulatory presumption, and evidence in the record. Specifically, Mrs. Norma J. Parsons is an eligible survivor under Act; Mr. Parsons had pneumoconiosis; and, his pneumoconiosis arose out of his coal mine employment. Consequently, the resolution of Mrs. Parsons' modification request involves an evaluation of the record to determine whether a mistake of fact occurred in Judge Neal's determination that Mr. Parsons' death was not caused by coal workers' pneumoconiosis.

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[47]20 C.F.R. § 718.4 indicates that the definitions in 20 C.F.R. § 725.101 are applicable. 20 C.F.R. § 725.101, in turn, refers to the term "survivor" as used in Subpart B of Part 725. 20 C.F.R. § 725.214 then sets out the spousal relationship requirements and 20 C.F.R. § 725.215 describes the dependency rules. According to § 725.214 (a) the spousal relationship exists if the relationship is a valid marriage under state law. Under § 725.215(a), a spouse is deemed dependent if she was residing with the miner at the time of his death.

[48]20 C.F.R. § 718.205 (a) (1) and see *Trumbo v. Reading Anthracite Co*., 17 B.L.R. 1-85 (1993).

[49]20 C.F.R. § 718.201 (a) (1) and (2).

[50]20 C.F.R. § 718.201 (b).

[51]20 C.F.R. §§ 718.203 (a) and 205 (a) (2).

[52]20 C.F.R. § 718.205 (a) (3).

Death Due to Pneumoconiosis

For a survivor claim filed on or after January 1, 1982, the Department of Labor regulations provide four means by which to establish that a coal miner's death was due to coal workers' pneumoconiosis:[53]

1. The miner had complicated pneumoconiosis;

2. Death was caused by pneumoconiosis;

3. Death was caused by complications of pneumoconiosis; or,

4. Pneumoconiosis was a substantially contributing cause or factor leading to the miner's death.

However, a survivor may not receive benefits if the coal miner's death was caused by traumatic injury, or the principal cause of death was a medical condition not related to pneumoconiosis, unless evidence establishes that pneumoconiosis was a substantially contributing cause of death.

Complicated Pneumoconiosis

As previously discussed, upon consideration of the entire medical record, including the probative autopsy/biopsy evidence, I determined Mr. Parsons did not have complicated pneumoconiosis. As a result, Mrs. Parsons cannot establish death due to pneumoconiosis under 20 C.F.R. § 718.304.

Death Caused by Pneumoconiosis

In the record before Judge Neal in 2001, none of the physicians concluded that coal workers' pneumoconiosis directly killed Mr. Parsons. Likewise, in the additional opinions submitted during the modification proceedings, including Dr. Molony's observations, no doctors states Mr. Parsons' death was directly caused by the coal workers' pneumoconiosis. As reported by Dr. Zayed on the death certification, the direct cause of death was ventricular fibrillation. Similarly, as Dr. Castle observed, Mr. Parsons did not have the severe degree of coal workers' pneumoconiosis that can cause death.

Death Caused by Complications of Pneumoconiosis

In the record before Judge Neal, Dr. Zayed indicated that the presence of coal workers' pneumoconiosis could cause respiratory arrest with could then lead to fatal ventricular fibrillation. The physician also indicated pneumoconiosis could be a "co-morbid" condition that could contribute to such a condition. In assessing the probative value of Dr. Zayed's opinion, I

[53] 20 C.F.R. §§ 718.205 (c) (1), (2), and (3), and 304.

concur with Judge Neal's affirmed observation that Dr. Zayed's causation opinion is not well reasoned. Significantly, Dr. Zayed failed to identify the specific objective medical and pathological evidence that would make his assertions applicable in Mr. Parsons' case. In particular, the treating physician did not address whether, as established by the pathology studies, the presence of mild coal workers' pneumoconiosis in Mr. Parsons' lungs would cause respiratory arrest or represent a "morbid" condition. Additionally, as noted by Dr. Naeye, no evidence exists to indicate that respiratory arrest precipitated Mr. Parsons' fatal ventricular fibrillation. Accordingly, Dr. Zayed's causation opinion has diminished probative value.

In contrast, Dr. Fino, Dr. Castle, Dr. Morgan, Dr. Naeye, Dr. Caffrey, Dr. Rosenberg, and Dr. Hippensteel, produced a consensus opinion that coal workers' pneumoconiosis did not play a role in Mr. Parsons' death. Due to the minimal amount of coal workers' pneumoconiosis found during the pathological examination of Mr. Parsons' lung tissue and considering that the preponderance of the radiographic and clinical record did not show the presence or effects of pneumoconiosis, these physicians concluded coal workers' pneumoconiosis did cause or contribute to Mr. Parsons' death. Additionally, Dr. Castle stressed the minimal amount of coal workers' pneumoconiosis present in Mr. Parsons' lung tissue would not cause cardiac arrhythmia. The probative, well documented and reasoned medical opinions of Dr. Fino, Dr. Castle, Dr. Morgan, Dr. Naeye, Dr. Caffrey, Dr. Rosenberg, and Dr. Hippensteel outweigh Dr. Zayed's less probative, contrary assessment, and establish that Mr. Parsons' death was not caused by complications of pneumoconiosis.

Pneumoconiosis Was a Substantially Contributing Cause Of, Or Hastened, Death

Even though neither pneumoconiosis nor its complications caused Mr. Parsons' death and he did not have complicated pneumoconiosis, Mrs. Parsons may still be entitled to survivor benefits if pneumoconiosis was a substantially contributing cause of her husband's death. Prior to publication of the new regulations, the U.S. Court of Appeals for the Fourth Circuit, like several other federal appellate circuits, interpreted "substantially contributing cause" to include a hastening of a miner's death in any way. *Shuff v. Cedar Coal Co.*, 967 F.2d 977, 980 (4th Cir. 1992) and *Richardson v. Director, OWCP*, 94 F.3d 164 (4th Cir. 1996). Adopting that standard, the new regulation, 20 C.F.R. § 718.205 (c) (5), states "pneumoconiosis is 'a substantially contributing cause' of a miner's death if it hastens the miner's death." Under this legal standard, if pneumoconiosis cut short Mr. Parsons' life in any manner, Mrs. Parsons may prevail with her modification request and survivor claim.

To support the present modification request, Mrs. Parsons has also presented the opinion of Dr. Molony who stated that the presence of pulmonary emphysema and pneumoconiosis was a contributing factor in Mr. Parsons' death. The probative value of Dr. Molony's contribution opinion is greatly diminished due to his failure to identify the objective medical evidence that supports his assessment. His assessment also suffers from the absence of any explanation for how he determined the mild coal workers' pneumoconiosis in Mr. Parsons' lungs contributed to his death. The mere presence of coal workers' pneumoconiosis is an insufficient basis to conclude that it contributed to death.

Again, in contrast, Dr. Fino, Dr. Castle, Dr. Morgan, Dr. Naeye, Dr. Caffrey, Dr. Rosenberg, and Dr. Hippensteel emphasized that the pathological evidence, coupled with the radiographic and clinical evidence, demonstrated the severity of Mr. Parsons' coal workers' pneumoconiosis was too minimal or mild to have played any role in his death. Integrating the arterial blood gas studies, Dr. Rosenberg also emphasized that Mr. Parsons' lung disease did not sufficiently interfere with the oxygenation of his blood to have contributed to his heart condition. The more probative consensus Dr. Fino, Dr. Castle, Dr. Morgan, Dr. Naeye, Dr. Caffrey, Dr. Rosenberg, and Dr. Hippensteel outweigh Dr. Molony's less probative, contributing factor conclusion and establishes that coal workers' pneumoconiosis was not a contributing factor to, and did not hasten, Mr. Parsons' death.

Summary

In her affirmed denial of Mrs. Parsons' survivor claim, Judge Neal concluded the preponderance of the more probative medical opinion failed to establish that Mr. Parsons' death was due to coal workers' pneumoconiosis. Upon consideration of the entire medical record, including the additional medical opinions presented during the modification proceedings, I find no mistake of fact occurred in Judge Neal's denial of Mrs. Parsons' survivor claim. The preponderance of the more probative medical opinion establishes that neither coal workers' pneumoconiosis nor its complications caused, contributed to, or hasten the death of Mr. Parsons.

CONCLUSION

The parties' stipulations, pathological evidence, and presumptions, have established that Mr. Parsons had coal workers' pneumoconiosis and suffered a totally disabling pulmonary. However, the preponderance of the more probative opinions by physicians who evaluated the entire medical record, including the pathological findings of mild, simple coal workers' pneumoconiosis, demonstrates that coal workers' pneumoconiosis did not cause or contribute to Mr. Parsons' total pulmonary impairment. As a result, the last requisite element of entitlement, total disability due to coal workers' pneumoconiosis, has not been proven. Accordingly, Mr. Parsons' claim for black lung disability benefits under the Act must be denied.

Upon consideration of the entire record, I find no mistake of fact occurred in Judge Neal's denial of Mrs. Parsons' survivor because the preponderance of the more probative medical opinion failed to establish that Mr. Parsons' death was due to coal workers' pneumoconiosis. Accordingly, Mrs. Parsons' modification request must be denied.

ORDER

The black lung disability claim of Mr. BILLY J. PARSONS is **DENIED**. The modification request to the survivor by MRS. NORMA J. PARSONS is **DENIED**.

SO ORDERED:

A

RICHARD T. STANSELL-GAMM
Administrative Law Judge

Date Signed: June 7, 2006
Washington, DC

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. See 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. See 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. See 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).

Attachment No. 1

American Board of Medical Specialties

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Attachment No. 2

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